EXHIBIT C26

REDACTED APPENDIX D

FILED UNDER SEAL SUBJECT TO DISCOVERY CONFIDENTIALITY ORDER DATED MARCH 1, 2017

CTEH®

EXPERT REPORT OF

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In Re: Johnson & Johnson Talcum Powder Products Marketing, Sales Practices And Products Liability Litigation

February 25, 2019

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List of Acronyms

ACGIH	American Conference of Governmental Hygienists
ACM	Asbestos-containing material
AHERA	Asbestos Hazard Emergency Response Act
AIHA	American Industrial Hygiene Association
ATSDR	Agency for Toxic Substances and Disease Registry
CAS	Chemical Abstracts Service
CIR	Cosmetic Ingredient Review
CTFA	Cosmetics Toiletries, and Fragrances Association
FDA	Food and Drug Administration
GHS	Globally Harmonized System
GRAS	Generally recognized as safe
HCS	Hazard Communication Standard
HSDB	Hazardous Substances Data Bank
IARC	International Agency for Research on Cancer
IFRA	International Fragrance Association
IRIS	Integrated Risk Information System
ISRTP	International Society of Regulatory Toxicology and Pharmacology
LOAEL	Lowest observed adverse effect level
MSDS	Material safety data sheet
MTD	Maximally tolerated dose
NCI	National Cancer Institute
NIOSH	National Institute for Occupational Safety and Health
NOAEL	No observed adverse effect level
NTP	National Toxicology Program
OECD	Organization for Economic Co-operation and Development
OSHA	Occupational Safety and Health Administration
PCM	Phase contrast microscopy
PCME	Phase contrast microscopy equivalent
PDQ	Physician Data Query
RoC	Report on Carcinogens
RTECS	Registry of Toxic Effects of Chemical Substances
SOT	Society of Toxicology
TEM	Transmission electron microscopy
TOXNET	Toxicology Data Network
TWA	Time weighted average
USEPA	United States Environmental Protection Agency

USP	United States Pharmacopeia
WHO	World Health Organization

1.0 Introduction & Qualifications

I am a Senior Toxicologist at the Center for Toxicology and Environmental Health, LLC (CTEH®), an environmental consulting firm that is associated with the University of Arkansas Medical Sciences BioVentures Program. CTEH® has several specialties, including toxicology, human health risk assessment, industrial hygiene, indoor air quality, and emergency response.

I hold a Bachelor of Science degree in Veterinary Science, Biomedical option from the University of Nebraska in Lincoln, Nebraska (2008) and a Ph.D. degree in Toxicology from Texas A&M University in College Station, Texas (2013). Since earning my Ph.D., I have been actively involved in the areas of toxicology, human health risk assessment, and emergency response. Since 2013, I have been active at CTEH® as a consultant in the area of human and environmental toxicology and have been involved in numerous projects involving the assessment of chemical exposure and their effects on humans. My current duties at CTEH® include serving as a consulting toxicologist; providing guidance for risk assessment and remediation plans; leading responses to, and providing toxicological support for, hazardous materials incidents; and providing toxicological support to care providers and workers with potential chemical exposures. As a toxicologist, I routinely assist in the determination of disease causation by evaluating chemical exposure and the scientific evidence relating exposure to human diseases according to the methodology of toxicological causation analysis. In addition, I am certified in the comprehensive practice of industrial hygiene by the American Board of Industrial Hygiene (CIH #11510CP).

I am a member of the Society of Toxicology (SOT), the Lone Star Chapter of the Society of Toxicology, the American Conference of Governmental Industrial Hygienists (ACGIH), and the American Industrial Hygiene Association (AIHA). I have written peer-reviewed publications and grants in toxicology and related fields. A list of my publications is included within my attached curriculum vitae (Appendix B). I have been retained in this case as an expert in Toxicology and Industrial Hygiene.

Toxicology, a blend of biology, chemistry, and medicine, is the science of the adverse effects of substances (e.g., chemicals, physical agents, drugs) on biological systems including the effects, the recognition, and the mechanisms of a chemical-related disease. Whether a substance is toxic depends upon two inseparable criteria: 1) the intrinsic nature of the substance, and 2) the dose, or how much of a substance the individual actually takes into his or her body. In toxicology, we study the dose-response of chemicals on biological systems, with emphasis on understanding the mechanisms of harmful effects. Toxicologists also provide expert opinions with respect to causation in toxic tort litigation. As stated by the Federal Reference Manual on Scientific Evidence (Federal Judicial Center, 2011), "*In tort litigation, toxicologists offer evidence that either supports or refutes plaintiffs' claims that their diseases or injuries were caused by chemical exposures.*"

2.0 Objective

I have been asked to assess the scientific literature regarding the alleged causal relationship between cosmetic talcum powder exposure and ovarian cancer, specifically with regard to the potential exposures to talc, asbestos, heavy metals and fragrant chemicals. I have also been asked to assess the scientific

methodology used by certain of the plaintiffs' experts in their assessment of the scientific literature and their independent research and opinions in their causal assessments.

The opinions stated in this report are based on my education, training, and experience in the field of toxicology and my review of the referenced information. All of my opinions in this report are stated to a reasonable degree of toxicological and scientific certainty.

CTEH® is compensated for my time at a rate of $305 per hour.

I reserve the right to amend this report as new information becomes available.

3.0 Materials Reviewed and Relied Upon

In formulating my opinions, I have relied upon and/or reviewed information from the following sources:

- Expert Report and Deposition of Dr. Crowley
- Expert Report and Deposition of Dr. Longo and Dr. Rigler
- Expert Report of Dr. Kane
- Expert Report of Dr. Alan Campion
- Expert Report of Dr. McTiernan
- Expert Report of Dr. Zambelli-Weiner
- Expert Report and Deposition of Dr. Carson
- Expert Report of Dr. Clarke-Pearson
- Expert Report of Dr. Kessler
- Expert Report of Dr. Smith
- Expert Report of Dr. Siemiatycki
- Expert Report of Dr. Wolf
- Expert Report and Deposition of Dr. Zelikoff
- Expert Report and Deposition of Dr. Plunkett
- Expert Report of Dr. Krekeler
- Expert Report of Dr. Moorman
- Expert Report of Dr. Smith-Bindman
- Expert Report of Dr. Cook
- Expert Report of Dr. Levy
- Expert Report of Dr. Singh
- Expert Report of Dr. Saed
- IMERYS and Johnson & Johnson Defendants' Documents cited in Dr. Cook and Dr. Krekeler Reports
- Exhibit 1 – Attorneys' Eyes Only – Johnson's Baby Powder Fragrance Ingredients
- Exhibit 2 – Attorneys' Eyes Only – Shower to Shower Fragrance Ingredients
- Exhibit 3 – Attorneys' Eyes Only – Changes to Johnson's Baby Powder Fragrance Ingredients
- JNJTALC000891091 – Formula Declaration Report
- JNJTALC000149667 – Formula Declaration Report
- Other provided documents

I have also reviewed the pertinent scientific literature. A list of this information is attached to this report (**Appendix A**). If additional information is made available, I reserve the right to supplement this report as necessary.

4.0 Summary of Conclusions

- The scientific literature regarding an association between talc use and ovarian cancer is varied and inconsistent. Studies that have found an association between ovarian cancer and talc use have only been able to establish a weak association, with hazard ratios well below 2.0, and the majority of studies have found no dose-response relationship. Additionally, the reliance of many of these studies on personal recall regarding talc use creates a potential for recall bias and makes it difficult to accurately account for the risk of misclassification of exposure.

- Application of the Hill Criteria to the scientific literature does not support the conclusion that there is likely a causal relationship between talc exposure and ovarian cancer.

- The cumulative asbestos exposures arising from the use of talcum powder products would fall within the background level of asbestos that every person living in the United States experiences, even accepting all of plaintiffs' experts' flawed methodologies for identifying asbestos. These levels of potential asbestos exposure would be of no causal significance for ovarian cancer, given that the International Agency for Research on Cancer (IARC) assessment of asbestos exposure and ovarian cancer and the relevant studies found an association with high levels of occupational exposure only.

- Even crediting plaintiffs' experts' claims to have found asbestos fibers in Johnson's Baby Powder and Shower to Shower, any hypothetical concentration of such asbestos in talcum powder would be <0.1%, and the product would therefore not be considered asbestos-containing.

- IARC's conclusions regarding the causal relationship between asbestos and ovarian cancer are a direct result of studies examining the heavy occupational exposure of women to asbestos products and fibers, not to consumer use of alleged asbestos-contaminated products, such as talcum powder. Exposure scenarios based on consumer use of talcum powder are drastically different from these high-level occupational exposures.

- None of the heavy metals listed by the plaintiffs' experts have been causally associated with ovarian cancer. The plaintiffs' experts opine that the classification of these heavy metals as probable, possible, or known human carcinogens for various other forms of cancer as discussed below implies that they must be carcinogenic to the ovary as well. However, it is not scientifically reliable to extrapolate from one type of cancer to another in this manner. Furthermore, the plaintiffs' experts fail to quantify the amounts of heavy metals present in talcum powder, and what levels would cause damage, if any, to the ovary.

- Heavy metals are needed for the normal and healthy function of the human body. The general population is exposed to all these metals on a regular basis through food, water, air, and other

sources. The levels of metals found in consumer products are below any levels that would be anticipated to present a hazard to the general public or a regulatory concern.

- None of the fragrant chemicals noted by plaintiffs' experts have been associated with ovarian cancer. Fragrant chemical components make up an extremely low percentage of cosmetic talcum powder products, a fact that is ignored by Dr. Crowley and the plaintiffs' experts who rely on his assessment. None of these chemicals is present in Johnson's Baby Powder or Shower to Shower at levels that would warrant a health or regulatory concern. Furthermore, none of the plaintiffs' experts are able to provide a dose of level of exposure to these substances that would cause damage, if any, to the ovary.

5.0 Scientific Causation Methodology

In order for adverse health effects to be attributed to a specific chemical exposure, a valid scientific causation analysis must be performed. Causation analysis is a two-phased process involving both general and specific causation.

General causation is the scientific determination as to whether or not the chemical or substance in question can cause a particular condition, symptom, or disease in the general population at some specified dose.

Specific causation is the scientific determination as to whether a known or alleged chemical/substance exposure an individual may have received was the most likely cause of the injuries, symptoms, or disease that the individual is experiencing or has, and to whether there are no potential alternate causes that could have resulted in the alleged injuries, symptoms or diseases.

The issue addressed in a general causation analysis is:

- Has the chemical(s) or substance in question been shown, at a sufficient dose, to cause the symptom(s), injuries or disease(s) in question in humans?

The methodology for proving general causation, as currently practiced, is a methodology that has undergone continual refinement for approximately the last 150 years. Probably the first effort to formalize this process occurred when scientists and physicians sought to establish the cause of diseases induced by infectious agents whose presence could not easily or readily be identified with the naked eye. (Evans, 1976, Yerushalmy and Palmer, 1959). A similar process was instituted in the 1960s as scientists began to focus on diseases induced by chemicals associated with the workplace, our environment, or lifestyle choices like smoking (USDHEW, 1964, Hill, 1965). Known as the Hill Criteria, these characteristics are to be considered when assessing whether the relationship between a substance and disease is causal (Hill, 1965). According to the Hill Criteria, the following aspects should be considered:

1. The *strength* of the association between the disease and the substance. An example used by Hill is the increased risk of lung cancer in cigarette smokers, noting that heavy cigarette smokers have a death rate from lung cancer that is 20 or 30 times greater than the general population.

2. The *consistency* of the observed association. This refers to whether the association has "been repeatedly observed by different persons, in different places, circumstances, and times" (Hill, 1965).

3. The *specificity* of the association. Hill states that "*if… the association is limited to specific workers and to particular sites and types of disease and there is no association between the work and other modes of dying, then clearly that is a strong argument in favour of causation"* (Hill, 1965).

4. The *temporality* of the association, referring to the timeline of exposure and disease (in the case of toxicology) (Hill, 1965).

5. The *biological gradient,* or *dose-response*, of the association. The presence of a clear dose-response in the case of assessing the causal association between disease and exposure according to Hill "*admits of a simple explanation and obviously puts the case in a clearer light"* (Hill, 1965).

6. The *plausibility*, specifically the *biological plausibility*, of the suspected causation (Hill, 1965).

7. The *coherence* of the causation – meaning that the cause and effect hypothesis "*should not seriously conflict with the general known facts of natural history and biology*" (Hill, 1965).

8. Does ex*perimental* evidence support the association? The example provided by Hill whether a preventative action based upon the observed association does in fact prevent?

9. *Analogy* – or comparing similar evidence from a similar drug or disease.

Hill concludes that these nine viewpoints should be studied to determine whether an association is the result of a causal relationship, noting that while none of these viewpoints provides "indisputable evidence," they help to answer the question of whether a causal relationship is the most likely explanation for an association (Hill, 1965).

It is well recognized within the scientific/medical community that the above criteria form the scientifically accepted method for establishing general causation. These or very similar criteria have been adopted by the World Health Organization (WHO) (WHO, 1987), the International Agency for Research on Cancer (IARC) (IARC, 2006), the United States Environmental Protection Agency (USEPA) (USEPA, 2005) and the American Conference of Governmental Industrial Hygienists (ACGIH) (ACGIH, 2016). Numerous respected epidemiological texts adhere to the Bradford Hill Criteria as the methodological basis for evaluating causality (Mausner and Kramer, 1985, Monson, 1990, Hernberg, 1992). Casarett and Doull's Toxicology textbook, which is commonly used to teach toxicology to undergraduate, graduate, and medical students, presents these causation criteria for evaluating epidemiological data (Faustman and Omenn, 2013). A number of occupational medicine textbooks (Schenker, 1997, Eisen and Wegman, 1995) describe these criteria as the method by which epidemiological evidence is evaluated. Finally, this methodology is described in the Reference Manual on Scientific Evidence as the factors that guide judgments about causation (Federal Judicial Center, 2011).

The fundamental issues of general causation are generally answered in the scientific literature; however, the fundamental nature of specific causation, i.e., whether the chemical or substance exposure in question caused a particular individual's health effects, cannot be answered only by the scientific literature. The mere fact that a chemical or substance may be capable of producing the health effects in question does not mean that a particular person's specific condition is a direct result of the chemical or substance exposure incident.

The process for establishing specific causation has been established in the scientific literature for many years (Sackett et al., 1991, Sullivan, 1992, Guzelian et al., 2005). Assuming that general causation has been established for a given chemical(s) and disease(s), the following additional steps are required to establish specific causation in an individual scenario:

1. The exposure was of sufficient magnitude (concentration and duration, or dose) to produce the alleged injury, symptom, or disease (**Satisfying the Principle of Dose-Response**).
2. The exposure was temporally related to the onset of the alleged medical condition, meaning it happened before the condition, with sufficient time between the exposure and onset of symptoms (**Satisfying the Principle of Temporality**).
3. Potential alternate causes of the medical condition (confounders) can be adequately ruled out (**Eliminating Alternative Possible Etiologies for the Condition**).
4. There is coherence and consistency in the evidence evaluated in this specific case (**Establishing that the Evidence is Consistent with all Scientific Facts and Beliefs**).

Using established accepted methodology is critical in developing causal opinions and separating mere associations from causal relationships. I note that some of plaintiffs' experts have attempted to blur this line. Dr. Smith-Bindman, for example, suggested that modifiable behaviors that are associated with increased risk of disease must be treated as causal. (Smith-Bindman Deposition Vol. I, pages 113-14.) But that is not correct; the literature might well identify certain behaviors as a "risk factor" for disease in the sense that the two are associated statistically, but as the National Cancer Institute explains, the hard work of proving that such an association reflects a true causal relationship must still be done:

> "*Most cancer risk … factors are initially identified in epidemiology studies. In these studies, scientists look at large groups of people and compare those who develop cancer with those who don't. These studies may show that the people who develop cancer are more or less likely to behave in certain ways or to be exposed to certain substances than those who do not develop cancer.*
>
> *Such studies, on their own, cannot prove that a behavior or substance causes cancer. For example, the finding could be a result of chance, or the true risk factor could be something other than the suspected risk factor...*
>
> *When many studies all point to a similar association between a potential risk factor and an increased risk of cancer, and when a possible mechanism exists that could explain how the risk factor could actually cause cancer, scientists can be more confident about the relationship between the two*" (NCI, 2015).

6.0 Toxicological Principles

6.1 Dose and Exposure Concentration

Toxicology is the science that studies the adverse effects of chemicals or physical agents (toxicants) on living organisms (Klaassen, 2013). A toxicant is any agent that can produce adverse effects on a biological system. This general definition of a toxicant highlights the fact that exposure to any substance can produce an adverse effect at a high enough dose. This fundamental principle of toxicology was coined by the Swiss physician and philosopher, Paracelsus, who stated:

> *"All substances are poisons; there is none which is not a poison. The right dose differentiates a poison from a remedy."* (Klaassen, 2013)

Toxicologists research and examine the mechanisms and functional effects of toxicants, as well as the probability of their occurrence. Generally speaking, the toxic effects of a substance are not produced unless that substance or its metabolic byproduct reaches the appropriate site in the body at a concentration and length of time that is sufficient to produce the effect (Klaassen, 2013). Thus, the toxic effect is not only dependent on the substance and physical nature of the toxin, but the exposure situation, how the toxin is metabolized, the concentration of the toxin at the target site, and the overall susceptibility of the subject.

The correlation between different dose levels/exposure concentrations and the effects they may elicit on an individual is referred to as the dose-response relationship and is integral to the process of characterizing toxicants. This relationship is considered the most fundamental concept in toxicology (Klaassen, 2013). The dose[1] and the dose-response relationship[2] are key concepts in the process of evaluating whether an exposure will result in an adverse health effect. Since all substances can be toxic, evaluation of exposure conditions (i.e., method of exposure) and dose levels (i.e., exposure science) to assess safety is vital for correctly estimating risk. An accurate assessment of toxicity of such substances depends on an understanding of the magnitude (i.e., exposure concentration) and duration of exposure to these substances of concern.

Toxicologists and epidemiologists were key players in the development of exposure and risk assessment methodologies, which incorporate information regarding the toxic properties of chemicals and contaminants (Federal Judicial Center, 2011). The National Research Council's Committee on the Institutional Means for Assessment of Health Risk stated that a human health risk assessment should include a *"description of the potential adverse health effects based on an evaluation of results of*

[1] The dose is defined as the amount of the chemical reaching the target organ being adversely affected by the chemical. It is less than the absorbed dose as the chemical is distributed throughout the body; the dose is a function of the blood level of a chemical reaching the organ and the organ's affinity and uptake of the chemical. It is the amount that actually produces the toxicity observed in the target organ.

[2] A fundamental principle of toxicology, often described mathematically, that states the extent of a response to an agent or substance at a specific dose. Where the dose is insufficient, then there will be no response. If a sufficient dose occurs, then the extent of the response will be proportionate to the dose, until at some dose and higher doses a maximal response is achieved.

epidemiologic, clinical, toxicological, and environmental research (hazard identification); extrapolation from those results to predict the type and estimate the extent of health effects in humans under given conditions of exposure (dose-response assessment); judgments regarding the number and characteristics of persons exposed at various intensities and durations (exposure assessment); summary judgments on the existence and overall magnitude of the public-health problem; and characterization of the uncertainties inherent in the process of inferring risk (risk characterization)" (Federal Judicial Center, 2011).

Controlled animal tests are often conducted to evaluate the toxicity of various substances. This type of testing is common in the field of toxicology, as controlled laboratory studies allow for the evaluation of toxic potency and an understanding of the dose-response relationship of substances and living organisms. The dose-response relationship reflects a substance's presence in an organ or tissue at high enough concentrations and for a long enough period of time to cause an adverse effect. Typically, a range of doses is administered to groups of animals in a study in order to capture the full range of responses, ranging from the No Observed Adverse Effects Level (NOAEL – the highest dose tested that does not elicit an adverse effect), the Lowest Observed Adverse Effects Level (LOAEL – the lowest dose tested that elicits an adverse effect), to a maximal effect or response (MTD – Maximally Tolerated Dose).

Animal models for ovarian cancer have been developed for nearly 40 years (Bobbs et al., 2015). There are a few species that develop ovarian tumors spontaneously, along with other models in which the animals are genetically modified so that exposure to environmental toxins can promote ovarian tumors (Vanderhyden et al., 2003). Spontaneous tumors have been shown to occur in laying hens, several strains of mice, and in rats in a wide variety of sub-types and at varying ages (Vanderhyden et al., 2003, Mullany and Richards, 2012). Other studies have also used non-human primates, which have similar reproductive characteristics to humans (King and Burdette, 2011). Numerous transgenic mice have also been developed, with different gene manipulations to increase their susceptibility to ovarian cancer (King and Burdette, 2011, Mullany and Richards, 2012, Bobbs et al., 2015). In addition to the spontaneous generation of ovarian cancer in various animal models, the transplantation of either ovarian surface epithelium or of ovarian cancer cells into immunosuppressed animals can also be used for animal models of ovarian cancer. The onset of ovary tumors has been examined in animals through multiple mechanisms, such as irradiation, chemical exposure, genetic defects and aging (Vanderhyden et al., 2003). Animal models of ovarian cancer where rodent ovaries were exposed to possible carcinogens date back to the 1970s (Shan and Liu, 2009).

The dose-response data from a toxicology or (sometimes) epidemiology study can be used to estimate the probability, or risk, that an adverse effect may occur, given a specific exposure level. In general, risk is proportional to both toxicity and exposure as follows:

$$\textbf{Risk} \approx \textbf{Toxicity} \times \textbf{Exposure}$$

It is important to note that dose-response data does not generally come from cell culture, or *in vitro*, testing. These experiments are useful to provide information about the mechanisms of toxicity, and they are more quickly performed and less expensive than animal or epidemiological or other studies (Klaassen, 2013). However, their relevance to whole body (*in vivo*) toxicity limits their use in decision-making without

the additional support of animal or epidemiological studies (NRC, 2007a, NRC, 2007b). For example, *in vitro* testing is unable to mirror the metabolism of a whole animal (NRC, 2007b). Extrapolation of a dose in *in vitro* testing to an environmental exposure would require modeling to estimate the environmental exposure, or human consumption, that would lead to the human tissue concentrations similar to those in the cell culture study. This requires physiologically based modeling and human data to provide information on background chemical exposures and disease processes (NRC, 2007b).

6.2 Risk Assessment Process

The risk assessment process is scientific in nature and dependent upon several factors: (1) how much of the substance is present in an environmental medium (e.g., soil, water, air); (2) how much contact (exposure) an individual has with the contaminated environmental medium; and (3) the inherent toxicity of the substance (USEPA, 2017). In order to conduct a risk assessment, a framework is necessary to evaluate information about chemical substances obtained from scientific studies and site environmental data to estimate a theoretical risk for individuals who may be exposed to the substances of interest. Risk assessment is built on the framework of four basic steps as recommended by the National Academy of Sciences, including: (1) hazard identification, (2) dose-response assessment, (3) exposure analysis, and (4) characterization of risk (NRC, 1983). The importance of the dose-response assessment and exposure analysis are described in detail below.

6.2.1 The Health-Protective Nature of Risk Assessment

Risk assessment is used in the United States and throughout the world as a tool for the systematic, scientific characterization of the potential for adverse health effects to occur. Risk assessment addresses human and ecological concerns based on the types of hazards, extent of exposure, and information regarding exposures and responses. Risk assessment is used by the Food and Drug Administration (FDA) to set safe levels of substances in food and drugs. It is also used by the National Institute for Occupational Safety and Health (NIOSH), the USEPA, the Occupational Safety and Health Administration (OSHA), ACGIH, IARC and other organizations to establish safe levels of substances in work settings, the environment, consumer products, and other aspects of everyday life. Risk assessment uses science to determine the likelihood of an effect from a potential exposure.

Human health risk assessment, as practiced by regulatory agencies, has been defined as "*the characterization of the potential adverse health effects of human exposures to environmental hazards*" (NRC, 1983).

Risk assessments provide regulatory agencies and risk managers with the ability to make informed decisions on hazardous site cleanup strategies that ensure overall protection of human health and the environment. However, a risk assessment is not intended to predict actual health effects that hazardous substances at a site may have on people. Instead, risk assessment provides a health protective estimation of the maximum risks potentially associated with a site. Regulators realize that uncertainty is inherent in the risk assessment process. In an effort to ensure that they err on the side of public health, many overestimated exposure assumptions are incorporated into the risk assessment process. This results in

health protective guidelines but does not provide an accurate depiction of the true human health risk. In other words, the guidelines typically overstate the true human health risk.

The risk assessment process is conservative on many levels. For example, the USEPA publishes toxicity values for hundreds of substances that serve as the toxicological underpinnings of regulatory human health risk assessments. These toxicity values are intended to protect even the most sensitive individuals in the general population and are derived by dividing the lowest dose associated with a toxic response in the most sensitive laboratory animal species by "uncertainty factors" designed to account for differences in responses between animals and humans and other sources of uncertainty in the model. Therefore, these toxicity constants are conservative (i.e. health protective) in nature and by design are likely to overestimate the hazard posed by a given dose of a substance.

Because risks are exaggerated to be protective and conservative in nature, human health risk assessment cannot be used to establish causation. It is useful to effectively rule out the possibility of health risks associated with a given exposure; however, it cannot be used to establish a causal effect. As noted by the California Environmental Protection Agency (2001),

> "*People sometimes think that a risk assessment will tell them whether a current health problem or symptom was caused by exposure to a chemical. This is not the case*" (CALEPA, 2001).

Due to its conservative nature (insofar as risks are exaggerated to provide increased margins of safety), risk assessment cannot be used to predict the incidence of health effects. However, since risk assessment provides upper bound risk estimates, it is particularly useful to effectively rule out the possibility of health risks associated with a given exposure. Thus, the methodology and guidance used by the USEPA provide a scientific foundation for the determination of health-protective constituent levels in the environmental media and provide a consistent basis for site assessment and risk-based decision-making regarding the corrective action process.

6.2.2 Calculating Dose and Exposure

The evaluation of exposure is a hallmark of industrial hygiene, toxicology, and risk assessment, as a hazard does not occur in the absence of exposure. Toxicologists regularly conduct exposure assessments to characterize the source, magnitude, frequency, route and duration of exposures to various agents (Klaassen, 2013, USEPA, 1992). The WHO defines exposure as the "*Contact of an organism with a chemical or physical agent, quantified as the amount of chemical available at the exchange boundaries of the organism and available for absorption*" (2001). Humans can be exposed to substances via skin contact (dermal), inhalation, or ingestion. An exposure can become an absorbed dose only when an agent crosses an absorption barrier (e.g., skin, lungs, digestive tract). Subsequent interactions of an absorbed dose with a target tissue can contribute to an adverse health outcome.

Although exposure to a hazardous substance is a requirement for risk, it is ultimately the dose that will relate to a toxic response. One can be exposed to a substance without receiving an internal dose, and an individual can be exposed to a substance that results in an internal dose insufficient to elicit a biological effect (USEPA, 2011). As stated in the Reference Manual on Scientific Evidence (Federal Judicial Center,

2011), "*Ultimately the dose incurred by populations or individuals is the measure needed by health experts to quantify risk of toxicity.*" Thus, the goal of an exposure assessment is to not only determine the type and amount of total exposure to a substance, but to identify specifically who was exposed and how large of a dose may be reaching target tissues.

6.2.3 Exposure Reconstruction

Exposure assessments are most often retrospective studies, in that the assessment is conducted after the exposure has occurred and generally after a disease or illness has been identified and reported. These situations require an exposure reconstruction to determine the scenarios where individuals were exposed to specific agents of concern, the time period of each exposure scenario, and the frequency, duration and intensity of each exposure.

Exposure assessment guidelines developed by the USEPA illustrate the fundamental steps of conducting an exposure reconstruction to evaluate historical exposures, be they occupational or of the general public (USEPA, 1992, Viet et al., 2008). The first step of any exposure reconstruction involves developing a study design that defines the scope of the reconstruction, including defining exposed/control populations, exposure time periods, locations, and the chemical agent(s) of interest. The study design process should involve a thorough literature review of past studies with relevant populations and exposures. The exposure reconstruction will ultimately assemble, extract and summarize records relevant to the exposure assessment, including work histories, exposure measurements and measurements of potential confounding exposures.

An exposure assessment is best suited for evaluating health risks when it is based upon quantified exposure measurements. A quantitative exposure assessment includes an estimation of the duration and intensity of an individual's potential exposure. Such data would include records of breathing-zone constituent concentrations or ambient air concentrations, exposure time, exposure frequency, and the number of years an individual was exposed. Exposure estimates may also be obtained from peer-reviewed literature, by interviewing exposed individuals, or with models that simulate the behavior of substances and predict their concentrations in the environment. When models are used, an investigator should attempt to collect site- or situation-specific data for the purpose of model validation (Williams et al., 2000). Furthermore, relying on subject interview data for an exposure reconstruction can introduce recall bias, namely when an individual's recollection of past exposures and behaviors is influenced by a belief that such exposures are associated with the individual's disease.

Quantitative exposure measurement data may not always be available, and in certain cases, it may only be possible to determine qualitatively whether or not an exposure may have taken place. A qualitative exposure assessment does not rely on quantitative exposure data and will therefore not provide the level of information (such as dose-response relationships) as quantitative or semi-quantitative assessments (Sullivan and Krieger, 2001, Viet et al., 2008). The results of a qualitative exposure assessment are generally not definitive and are expressed in terms of the '*probability*' of exposure being high, medium, low or none. Although a qualitative assessment can be used to determine whether individuals may have potentially been exposed to the agent of concern (i.e., ever/never exposed), it cannot establish that individuals received a dose sufficient to cause a disease. A qualitative exposure assessment is therefore

not useful for establishing causation or estimating an individual's lifetime health risks from exposures to a particular agent.

6.3 Genotoxicity

Genotoxicity is a genetic term that refers to a destructive effect on a cell's DNA or RNA. Genotoxicity generally falls into three categories: 1) carcinogens, or cancer-causing, 2) mutagens, or mutation causing, and 3) teratogens, or causing birth defects (Shah, 2012). Additionally, genotoxins can affect either germ cells (i.e., sperm or egg cells) or somatic cells (adult cells).

In germ cells, genotoxicity is associated with genetic diseases such as cystic fibrosis, Down Syndrome, or Tay-Sachs disease. Genotoxins in germ cells may even lead to spontaneous abortions or reduced fertility (Wurgler and Kramers, 1992). General Mendelian genetic characteristics play a role in genotoxicity depending on whether dominant or recessive genes are impacted by the genotoxin in germ cells. In somatic cells, various forms of genotoxicity may also turn off or on various genes in the cell (Klaassen, 2013). In the role of carcinogenesis, which will be discussed more in the subsequent section, this may include the activation of an oncogene, or the inactivation of a tumor-suppressor gene. Additionally, genotoxins may induce alterations that are related to other degenerative diseases such as cardiovascular disease, autoimmune defects, diabetes, and other diseases (Wurgler and Kramers, 1992). Thus, the fact that a material is genotoxic is not sufficient to also classify the material as carcinogenic. The two classifications are not mutually exclusive; nor are they mutually inclusive. For example, there are compounds that are considered carcinogens that are not genotoxic, such as estrogen (17β-estradiol), 1,4-dichlorobenzene, arsenic, and others (Hernandez et al., 2009). Comparatively, compounds may also be genotoxic but not a carcinogen, such as sodium azide, a compound used in automobile air bags and as a biocide in agriculture, which was genotoxic in assays but was not found to cause cancer (CDC, 1991).

Not only are the types of genetic changes caused by genotoxins diverse, but they also have varying effects on the cell depending on where the genetic change occurs. Some changes may result in gene mutations while others result in chromosome alterations. Additionally, many different assays exist for assessing genotoxicity, using bacteria, human cells, fungi, plants, animals, and germ cells (Klaassen, 2013). As with other toxicology studies, dose plays a key role in assessing the genotoxicity of a compound. An example of this is grain alcohol, or ethanol. Ethanol is a known human carcinogen and genotoxin, and a review of the Registry of Toxic Effects of Chemical Substances (RTECS) database and Toxicology Data Network (TOXNET) for information shows a multitude of genotoxic tests with varying doses and endpoints.

6.4 Carcinogenicity

Carcinogenicity is a chemical or compound's ability to cause cancer in living tissue. The onset of cancer is a multistep process, meaning that multiple steps must follow a temporal sequence to promote the uncontrolled growth of living tissue (Klaassen, 2013).

Initiation is the first stage of the onset of cancer, and is normally defined as a "*rapid, irreversible process that results in a carcinogen-induced mutational event"* (Klaassen, 2013). There are multiple ways that this damage can occur, such as DNA modification, mutation and genotoxicity (as described above). However,

it is important to note that initiation alone is not sufficient to cause cancer. Once a cell has been initiated there are three potential outcomes: 1) the cell remains non-dividing through the natural regulatory processes of the body; 2) the cell is killed through either a terminal mutation or through the natural defense mechanisms of the body; or 3) the cell undergoes division that results in the growth and proliferation of the cell.

After a cell in the body has been initiated, the second stage of carcinogenesis is *promotion*. Promotion is defined as the "*endogenous or exogenous stimuli of cell growth*" (Klaassen, 2013). Agents that promote growth are not mutagenic and generally cannot induce tumors by themselves, but rather promote continued cell proliferation, inhibit cell death (apoptosis), and otherwise produce effects that are generally reversible, such as inflammation. For promotion to occur, multiple exposures/treatments or prolonged exposures/treatments are usually necessary, and these agents demonstrate a threshold for their effects. The term "co-carcinogen" is typically applied to promoters (Potter, 1980, Slaga et al., 1979). However, the definition of co-carcinogen can vary in the scientific literature. Bohrman states that a co-carcinogen may be carcinogenic, can be administered with a carcinogen or a promoter, requires long and repeated exposures, and has a probable threshold, whereas a promoter is not carcinogenic, must be given after an initiator, and also has a probable threshold requiring long and repeated exposures (Bohrman, 1983). In the case of inflammation as a promoter, the scientific literature generally defines inflammation as a "*response of living tissue to **local** injury…. That it leads to the **local** accumulation of blood cells and fluid*" (emphasis mine) (Ryan and Majno, 1977). The inflammatory response is directed to heal affected tissue, which involves direct interaction with this area, rather than a generalized response in distant tissues (Coussens and Werb, 2002). As with any promoter, the inflammation itself does not cause cancer, but rather promotes cell growth, requiring multiple prolonged exposures of a sufficient threshold.

The final stage of the induction of cancer is known as *progression*, or the alteration of pre-neoplastic lesions (benign) into cancer (Klaassen, 2013). During this phase, the increased proliferation of the cells causes an increase in DNA synthesis, which allows for additional damage to the DNA, including chromosomal changes, translocations, mutations, and epigenetic changes. These additional changes cause the cells to outgrow the surrounding "normal" cells and to attract all the nutrients needed for sustained growth. Progression, similar to initiation, is an irreversible event, and while it may occur as a result of exposure to carcinogens, it may also occur spontaneously as a result of the increased proliferation of cells.

6.4.1 Classification of carcinogens

Several agencies assess the evidence of carcinogenic risks to humans and have derived criteria and classifications based upon the state of the scientific research. These agencies include IARC, USEPA, and the National Toxicology Program (NTP). It is important when examining carcinogenicity data to understand how the different agencies define and classify chemicals and compounds as carcinogens.

6.4.1.1 International Agency for Research on Cancer (IARC)

The IARC Classification includes carcinogenicity in humans and carcinogenicity in animals. Carcinogenicity in humans based on appropriate evidence is classified into the following:

- **Sufficient Evidence of Carcinogenicity.** Defined by IARC as "*a positive relationship has been observed between the exposure and cancer in studies in which chance, bias, and confounding could be ruled out with reasonable confidence* (IARC, 2006)." IARC then notes that a separate sentence then identifies the target organ(s) or tissue(s) where an increased risk of cancer has been observed in humans.

- **Limited Evidence of Carcinogenicity.** Defined by IARC as "*a positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered … to be credible, but chance, bias, or confounding could not be ruled out with reasonable confidence* (IARC, 2006)."

- **Inadequate Evidence of Carcinogenicity.** Defined by IARC as "*[t]he available studies are of insufficient quality, consistency, or statistical power to permit a conclusion regarding the presence or absence of a causal association … or no data on cancer in humans are available* (IARC, 2006)."

- **Evidence Suggesting Lack of Carcinogenicity.** Defined by IARC as "*There are several adequate studies covering the full range of levels of exposure that humans are known to encounter, which are mutually consistent in not showing a positive association between exposure to the agent and any studied cancer* (IARC, 2006)."

IARC also uses the following categories regarding the evidence of carcinogenicity in experimental animals:

- **Sufficient Evidence of Carcinogenicity.** Defined by IARC as "*a causal relationship has been established between the agent and an increased incidence of malignant neoplasms … in (a) two or more species of animals or (b) two or more independent studies in one species*" (IARC, 2006).

- **Limited Evidence of Carcinogenicity.** Defined by IARC as "[*t*]*he data suggest a carcinogenic effect but are limited for making a definitive evaluation"* for various reasons (IARC, 2006).

- **Inadequate Evidence of Carcinogenicity.** Defined by IARC as "[*t*]*he studies cannot be interpreted as showing either the presence or absence of a carcinogenic effect because of major qualitative or quantitative limitations, or no data on cancer in experimental animals are available"* (IARC, 2006).

- **Evidence Suggesting Lack of Carcinogenicity.** Defined by IARC as "[*a*]*dequate studies involving at least two species are available which show that … the agent is not carcinogenic"* (IARC, 2006).

Based upon the body of evidence in humans, animals, and in mechanistic studies, IARC then reaches an overall evaluation of the carcinogenicity of the agent to humans. The overall evaluations are categorized as:

Group 1: The agent is carcinogenic to humans

IARC uses this category when there is *sufficient evidence of carcinogenicity* in humans and may use this category when the human evidence is less than sufficient, but there is *sufficient evidence in animals*, and strong evidence that the agent acts through similar mechanisms in both experimental animals and humans.

Examples of agents classified by IARC as Group 1 carcinogens include alcoholic beverages (ethanol), diesel exhaust, menopausal hormone therapy, birth control, leather dust, processed meat, salted fish, and tanning beds (IARC, 2018).

Group 2A: The agent is probably carcinogenic to humans

IARC generally uses this category when there is *limited evidence of carcinogenicity* in humans and *sufficient evidence of carcinogenicity* in experimental animals. Sometimes IARC may use this category solely based upon a classification of *limited evidence of carcinogenicity* in humans or in other circumstances.

Examples of agents classified by IARC as Group 2a agents include anabolic steroids, glass manufacturing, burning wood or biomass fuel, working as a hairdresser or barber, red meat, shiftwork, and very hot beverages (IARC, 2018).

Group 2B: The agent is possibly carcinogenic to humans

This category is generally used when there is *limited evidence of carcinogenicity* in humans and less than *sufficient evidence of carcinogenicity* in experimental animals. An agent may also be classified as a Group 2b based on strong evidence from mechanistic or other relevant data.

Examples of agents classified as Group 2B by IARC include certain extracts, talc-based body powder, ginkgo biloba, aloe vera, working as a carpenter, pickled vegetables, and progesterone-only contraceptives (IARC, 2018).

Group 3: The agent is not classifiable as to its carcinogenicity

IARC generally uses this category for compounds for which there is *inadequate evidence* in humans and *inadequate* or *limited* evidence in experimental animals. This category is also used for compounds that do not fall into any other category, or for agents whose mechanism in experimental animals does not operate in humans.

Agents classified as Group 3 by IARC include cholesterol, caffeine, vitamin K, limonene, hair dye, silicone breast implants, and tea (IARC, 2018).

Group 4: The agent is probably not carcinogenic to humans

This category is used by IARC for agents for which there is *evidence suggesting a lack of carcinogenicity* in both humans and in experimental animals. In some instances, agents with *inadequate evidence* in humans are classified in this group based upon a range of mechanistic and other relevant data.

While 1,079 agents have been assessed by IARC, only one has been classified Group 4: caprolactam (IARC, 2018).

6.4.1.2 United States Environmental Protection Agency (USEPA)

The USEPA follows a weight of evidence procedure when assessing a compound's potential carcinogenicity (USEPA, 2005). Five recommended carcinogenicity rankings are used by the USEPA:

1. **Carcinogenic to Humans.** This classification "*indicates strong evidence of human carcinogenicity*" (USEPA, 2005). Various combinations of scientific evidence may fall under this classification, including "*convincing epidemiological evidence of a causal association between human exposure and cancer*", or "*a lesser weight of epidemiologic evidence that is strengthened by other lines of evidence*" such as extensive evidence in animal studies, including mode of action evidence (USEPA, 2005).

2. **Likely to Be Carcinogenic to Humans.** The USEPA defines this classification for when "*the weight of the evidence is adequate to demonstrate carcinogenic potential to humans but does not reach the weight of evidence for the description 'Carcinogenic to Humans'*" (USEPA, 2005). This classification covers a wide range of scientific evidence, which includes demonstrating a plausible (not causal) association between human exposure and cancer, with some supporting biological evidence, though not necessarily from animal studies. Another example of scientific evidence falling under this category is multi-species animal studies, with or without evidence of carcinogenicity in humans, or a positive tumor study in animals that is strengthened by other lines of evidence.

3. **Suggestive Evidence of Carcinogenic Potential.** The USEPA uses this carcinogenic category when the "*weight of evidence is suggestive of carcinogenicity; a concern for potential carcinogenic effects in humans is raised, but that data are judged not sufficient for a stronger conclusion*" (USEPA, 2005). This include scientific evidence of varying levels of concern, ranging from a single positive cancer study to a single positive study with extensive negative studies in other species.

4. **Inadequate Information to Assess Carcinogenic Potential.** This classification is used when "*available data are judged inadequate for applying one of the other descriptors*" (USEPA, 2005). Under this classification, additional studies would be anticipated to provide additional insight, and includes scientific evidence that has little or no relevant information, conflicting evidence, or negative evidence that is not sufficiently strong.

5. **Not Likely to Be Carcinogenic to Humans.** The USEPA uses this classification when "*available data are considered robust for deciding that there is no basis for human hazard concern*" (USEPA, 2005). This classification may be based upon various scientific data, including positive animal studies where there is strong evidence that the mode of action does not operate in humans, negative evidence in both humans and animals, and other lines of evidence as well.

6.4.1.3 National Toxicology Program (NTP)

Compared to the IARC and USEPA classifications above, the NTP uses a more simplistic categorization of carcinogenicity. As part of the Public Health Service Act, the NTP maintains a Report on Carcinogens (RoC) which uses the following two classifications:

1. **Known to Be a Human Carcinogen.** The NTP uses this classification when "*[t]here is sufficient evidence of carcinogenicity from studies in humans, which indicates a causal relationship*" (DHHS, 2016).

In the 14th Report on Carcinogens (RoC), the NTP classifies approximately 62 compounds as known human carcinogens, including compounds such as wood dust, sunlamps or sunbeds, and estrogens.

2. **Reasonably Anticipated to Be a Human Carcinogen.** This classification is used when "[*t]here is limited evidence of carcinogenicity from studies in humans … but that alternative explanations … could not adequately be excluded"* (DHHS, 2016). This classification may also be used when there is "*sufficient evidence of carcinogenicity from studies in experimental animals"* or, if there is less than sufficient evidence in animals and humans, if the agent belongs to a "*well-defined, structurally related class of substances whose members are listed in a previous Report on Carcinogens"* (DHHS, 2016).

In the 14th RoC, the NTP classified approximately 186 compounds as reasonably anticipated to be human carcinogens, including certain dyes, ultraviolet radiation and progesterone.

7.0 Talc

Broadly, "talc" can refer to mineral (i.e. pure) forms as well as a variety of industrial mineral products, which may be comprised of a range of pure talc in addition to other materials. Soapstone, talcite, steatite, and pyrophyllite are example metamorphic materials, which, varying in composition, all include talc to some degree. Talc was initially mined by the ancient Greeks and continues to be used today in a wide variety of products and industrial processes (Hildick-Smith, 1976). Pure talc is defined as a hydrous magnesium silicate, meaning that it is composed of water, magnesium, and silica and is characterized by hexagonal arrangements of planed sheets (a sheet containing magnesium ions sandwiched between two silica sheets) held together by weak forces, facilitating the lubricating attributes of the material.

Talc is used in a variety of industries, including agriculture, wastewater treatment, and the production of paper, rubber, ceramics and plastics. Exposure to talc can occur under occupational (mining, milling, fabrication) or consumer settings. NIOSH has established an 8-hour time weighted average (TWA) for respirable talc containing no asbestos fibers as 2 mg/m^3 (NIOSH, 2018). Cosmetic talc is generally used as a face powder or body powder by both adults and children, primarily to lubricate the skin and prevent chafing. Talc has been used cosmetically for hundreds of years. Cosmetic talc, or "talcum powder," normally contains 98% talc, while talc destined for pharmaceutical applications contains >99% talc (Zazenski et al., 1995). Comparatively, industrial talcs may have varied mineral composition, including other minerals such as silica or asbestos (Hildick-Smith, 1976). This is important when assessing studies of talc exposure in the scientific literature, as industrial talcs may have significantly different mineral compositions than cosmetic or pharmaceutical talc products.

7.1 Animal Models of Talc Exposure and Toxicity

In animal models of exposure, information regarding the mineralogy of the substance, fiber content, or particle size of the administered "talc" is often limited. The toxicity of talc has been investigated through a wide variety of exposure paradigms, many of which were aimed at determining the potential carcinogenicity of exposure. Oral exposures in rats to commercial talcs found no difference in tumor incidence (IARC, 2010). Inhalation studies in rodents using commercial or high purity talcs also found no

increase in lung tumors. One study found an increase in lung cancer when exposed to high levels of high-purity talc; however, the strain of rats used in this study was shown to have a high incidence of these tumors, and the trend was only seen in one lab, leading reviewers to conclude that the increase was not related to talc exposure (NTP, 1993, IARC, 2010). Subcutaneous injections of talc of an unknown type in female mice also showed no tumor incidence. Intraperitoneal injection of United States Pharmacopeia (USP) talc in two studies showed no mesotheliomas or other neoplasms; however, a study of peritoneal injection of commercial talc resulted in peritoneal mesotheliomas in both the treated and control groups, showing no specific trends. Intraperitoneal injections of a granular talc (type unspecified) resulted in one mesothelioma in the exposed rats, and another study of peritoneal injection of USP talc found three non-mesothelioma cancers in the exposed population compared with none of the controls (Pott et al., 1974, IARC, 2010).

One study examined the intrabursal injection of Italian talc, which was described as asbestos free with no additional characterization, in female rats. No neoplasms were reported, although the authors noted papillary changes in the treated animals. Other groups of animals were also assessed, but the results were not reported (Hamilton et al., 1984, IARC, 2010). Intravaginal applications of talc in rabbits or monkeys was not associated with translocation to the ovaries (Phillips et al., 1978, Wehner et al., 1985, Wehner and Weller, 1986, IARC, 2010). To study translocation of talc to the ovaries in rats from the NTP whole body talc exposure study (NTP, 1993), ten female rats were randomly selected from control and treatment groups for examination for the presence of material consistent with talc particles. No material consistent with talc was found in the ovaries or ovarian bursa from any of the rats examined, and there were no "*exposure-related lesions*" found in the ovaries of the rats either (Boorman and Seely, 1995).

The investigation of chronic toxicity has associated most histopathologies with intravenous, intrasplenic or intrapleural injections of talc (Eger and Canaliss, 1964, Dogra et al., 1977, IARC, 2010). Models of chronic inhalation of talc in rats and guinea pigs were mostly associated with inflammation, respiratory metaplasia and fibrosis (Wehner et al., 1977, NTP, 1993, IARC, 2010).

7.2 Occupational Talc Exposure

In humans, the outcomes associated with talc exposure in an occupational setting have been studied via epidemiological investigation. Cohort or case-control studies compare populations of individuals, consider their history with talc, and apply statistical methodologies to estimate the relative risk of developing certain diseases. It is crucial to acknowledge that the results from these studies present associative evidence only and do not provide any insight into causation. These studies must be interpreted with all potential confounding factors in mind. Although investigators attempt to control and account for as many covariates as possible, the retrospective nature of the studies means they are often subject to bias.

7.2.1 Occupational Exposure and Respiratory Disease

Six cohort studies focused on workers within the cosmetic talc mining and milling industry used death records to calculate the standardized mortality ratio (SMR) between workers and a reference population, typically the national death rates. These cohort studies failed to provide evidence to suggest that these job tasks were associated with an increase in mortality due to cancer (Rubino et al., 1976, Selevan et al.,

1979, Wergeland et al., 1990, Wild et al., 2002, Coggiola et al., 2003). A 1976 study, later updated in 1979, reported an excess mortality attributed to pneumoconiosis that increased with increasing exposure but reported no excess in the incidence in lung cancer (Rubino et al., 1976, Rubino et al., 1979). Two studies reported an excess in lung cancer mortality and non-malignant respiratory disease among some cosmetic talc workers, with both studies noting the potential for exposure to occupational hazards other than talc (Selevan et al., 1979, Wild et al., 2002). Another cohort study, updated in 2017, reported no excess in total cancer or lung cancer mortality and noted a significant increase in mortality from respiratory tract diseases, primarily attributed to silicosis (Pira et al., 2017, Coggiola et al., 2003). More recently, a cosmetic talc worker cohort was followed-up on to report a mild, non-significant increase in lung cancer and no excess mortality from non-malignant respiratory disease (Wergeland et al., 2017). Of the studies that reported it, there were no instances of mesothelioma in talc miners or millers (Coggiola et al., 2003, Pira et al., 2017, Wergeland et al., 2017, Wild et al., 2002).

Cohort studies also exist for workers in industries that deal with talc products, though there is typically little to no information about the type of talc used, thus making them less informative. These studies include workers in the manufacture of ceramic plumbing fixtures (Thomas and Stewart, 1987), the manufacture of pulp and paper (Langseth and Andersen, 1999, Langseth and Kjaerheim, 2004), and rubber manufacturing industries (Straif et al., 2000, Straif et al., 1999). In all cases where an excess of cancer mortality was determined, job tasks were associated with additional exposures to other occupational hazards (respirable silica, microbes, formaldehyde, asbestos, nitrosamines, or carbon black).

7.2.2 Occupational Exposure and Ovarian Cancer

Two studies examined the association between occupational exposures to talc and ovarian cancer (Hartge and Stewart, 1994, Chen et al., 1992). Neither study found an increased risk of ovarian cancer with occupational exposure to talc, although there were issues with both studies, including sample size, cancer-reporting issues, and selection bias.

7.3 Consumer Exposure to Cosmetic Talc and Ovarian Cancer

One of the earliest references to concerns regarding talc and ovarian cancer came in 1979 when Longo (not the Dr. Longo involved in this litigation) and Young published a paper regarding the use of cosmetic talc and ovarian cancer in response to a 1977 *Lancet* article that discussed cosmetic talc powder (Longo and Young, 1979). The authors concluded that the "*risk of cosmetic talc has not been fully evaluated*" and recommended that more intense research be performed to assess the potential relationship between cosmetic talc use and the risk of ovarian cancer.

7.3.1 Case-Control and Cohort Studies

Case-control and cohort studies are the predominant methods by which investigators can investigate the cosmetic use of talc for any increases in risks of developing ovarian cancer, the prevalent cancer alleged to be associated with regular use (IARC, 2010). In a review of case-control and cohort studies ranging in quality of data, there is some discordance of results as discussed below. Additional factors commonly accounted for were medical, sexual, and reproductive histories, methods of application, or the influence of sterilization procedures preceding or following talc use.

Perineal talc use was examined in 215 women with ovarian cancer and controls in a 1982 population-based case-control study that found a statistically significant increased risk of ovarian cancer associated with the use of talc for any perineal use (as a dusting powder or on sanitary napkins) (RR: 1.92) (Cramer et al., 1982). The authors did not find a statistically significant association with the use of talc as a dusting powder alone. Furthermore, no information on the duration or frequency of talc use was reported, there was a low rate of participation among the matched controls, and parity and menopausal status were the only confounders controlled for.

A reply to Cramer's study found no statistically significant association between genital talc exposure and ovarian cancer risk (RR: 2.5, CI 0.7-10.0) (Hartge et al., 1983). In this hospital-based case-control study, questions on potential talc exposure were not added until after the study began, and no information on duration or frequency of exposure was reported. The authors also did not control for potential confounders.

Another hospital-based case-control study found no statistically significant association between genital talc usage and ovarian cancer. Although the authors noted a trend with increasing frequency, none was evident with duration of exposure (Whittemore et al., 1988).

A 1989 hospital-based case-control study for ovarian cancer risk factors found that women who used talc on a weekly basis had an increased risk of ovarian cancer; however, there was no consistent trend of increasing risk with increasing frequency of talc use, and no statistical increase was seen with daily use of talcum products (Booth et al., 1989). The authors also note that there was no significant difference between the percentages of cases and controls who had used diaphragms that were kept in talc. This study failed to provide the participation rates, and questions regarding talc use were not added until several months into the study. Thus, potential talc exposure information was missing in approximately 18 cases and 17 controls. The authors also noted the potential for recall bias, and that participants were not asked the length of time that they used talc.

A 1989 population-based case-control study in western Washington State found an increased risk of ovarian cancer with the use of deodorizing talc; however, the study did not find an increased risk of ovarian cancer with use of baby powder or unspecified talc products (Harlow and Weiss, 1989). The study had limited information on powder use, and the study was small in size. Furthermore, there was no statistically significant association between any method of powder use (diaphragm storage, after bathing, on sanitary napkins, or various combinations of these methods) and risk of ovarian cancer.

A retrospective population-based case-control study investigating the risk factors for ovarian cancer was conducted in 1992 in 112 women with ovarian cancer and 224 controls in Beijing (Chen et al., 1992). In the study, 5 out of 224 women reported using dusting powder on their lower abdomen and perineum. 7 out of 112 women diagnosed with ovarian cancer reported using dusting powder on their lower abdomen and perineum, which the authors stated represents an increased risk of ovarian cancer (RR-3.9, CI 0.9-10.6). However, this increased risk was not statistically significant, and occupational exposure to talc (which was also evaluated) did not show an increased risk of ovarian cancer (RR-0.9, CI 0.3-2.9). No additional information was reported regarding frequency of dusting powder application, brand of dusting

powder, method of application, or age range of cases and controls, and the size of the study limits the study's conclusions.

An additional population-based case-control study in 1992 found an association between any perineal talc exposure (OR-1.5, CI 1.0-2.1), current talc use, daily talc use, or talc use longer than 10 years and a mildly increased risk of ovarian cancer. The authors found an increased risk of ovarian cancer with increasing frequency of talc applications, showing that the risk was greatest in women who applied talc at least once per day (OR-1.8, CI 1.1-3.0). The authors of this study suggest that the association between genital use of talc and risk of ovarian cancer is weak and it is unlikely that the exposure-disease pathway is the principal one involved in ovarian cancer (Harlow et al., 1992). In this study, one of the major difficulties was subject recall, and a potential confounder was the use of oral contraceptives, which were not accounted for.

A hospital-based case-control study found a statistically significant increase in ovarian cancer with sanitary napkin use with talc exposure (RR-4.8, CI 1.3-17.8); however, there was no statistically significant association between genital talc use and ovarian cancer (RR-1.7, CI 0.7-3.9) (Rosenblatt et al., 1992). In this study, the authors struggled to find appropriate controls for comparison, had a small study size, and limited information regarding perineal exposures to talc.

Another hospital-based case-control study assessed 189 women who underwent ovarian cancer surgery (Tzonou et al., 1993) and found no statistically significant association between perineal talc use and ovarian cancer. The study was somewhat limited by the low prevalence of perineal talc use in the study group.

Another population-based case-control study in Australia also found a "slight increase" in ovarian cancer with perineal talc use around the abdomen or perineum (OR-1.27, CI 1.04-1.54 (Purdie et al., 1995). Information regarding the frequency and duration of talc exposure was not reported. A population-based case-control study performed the next year found an increased association between ovarian cancer and moderate to high levels of talc use; however, the authors did not provide confidence intervals for talc use, blocking further interpretation regarding statistical significance (Shushan et al., 1996). However, the authors did not assess potential confounders such as parity, contraceptive use, menarche or menopause. Furthermore, the authors reported little information on talc use.

Another population-based case-control study examining the various risks associated with ovarian cancer found that patients with ovarian cancer were more likely to have used talcum powder products compared to controls (OR 1.6, CI 1.2-2.1) (Cramer and Xu, 1995). Frequency, duration and method of talc product use were not reported by the authors. Additionally, the authors included borderline malignant ovarian tumors in the case population and noted the potential for recall bias. The primary objective of this study was to examine the protective nature of tubal ligation or other surgery in the prevention of ovarian cancer.

A 1997 Canadian population-based case-control study found a moderately increased risk of ovarian cancer (OR 1.42, CI 1.08-1.86) with any exposure to talc with a borderline association with the duration of talc exposure and no association between frequency of exposure and risk (Chang and Risch, 1997). It is unclear in this study how cases were identified (i.e., cancer registry).

A population-based case-control study of the same year examined women in three counties of western Washington and found an increased association between ovarian cancer and use of genital talcum powder or use of genital deodorant spray (RR 1.6, CI 1.1-2.3 and RR 1.9, CI 1.1-3.1 respectively) (Cook et al., 1997). The authors note that a sizable number of women eligible for their study did not participate, that substantial differences in powder use between participating and nonparticipating women would over- or underestimate the true risks of ovarian cancer, and that powder brands were not assessed. The authors ultimately concluded that a history of perineal dusting or use of genital deodorant sprays resulted in a "modest influence" on the development of epithelial ovarian tumors (Cook et al., 1997). No association was found between cumulative lifetime perineal dusting and risk of ovarian cancer.

Another hospital-based case-control study compared women diagnosed with peritoneal cancer to women diagnosed with ovarian cancer and found an increased association between ovarian cancer and perineal talc use; however, there was a large difference between sample size and the authors noted the potential for recall bias (Eltabbakh et al., 1998). The authors also did not adjust for confounders such as menarche, menopause and parity, nor were healthy controls enrolled in the study.

A 1998 population-based case-control study examined French-Canadian women who had been diagnosed with ovarian cancer to identify risk factors associated with their disease (Godard et al., 1998). The authors found significant associations between family history, age at use of oral contraceptives, and age at childbirth, but did not find a statistically significant association between talcum powder use and ovarian cancer.

A population study in 1999 examined women in Massachusetts and New Hampshire and found an increased association between genital talc use and ovarian cancer (OR 1.60, CI 1.18-2.15) after normalizing for age, location, family history, parity, oral contraceptive use and BMI (Cramer et al., 1999). Interestingly, the authors found no significant association between perineal dusting of talcum powder, sanitary napkin dusting, or underwear dusting, but only found a significant association when incorporating multiple uses in the genital area. Also, the association between ovarian cancer and talcum powder use was not associated with the frequency of use, with only the shortest frequency (<30 uses per month) achieving statistical significance (Cramer et al., 1999). There was also no association between years of use or total applications of talcum powder. Another hospital-based case-control study that same year found no correlation between use of talcum powder and the risk of development of ovarian cancer, even with prolonged exposure (Wong et al., 1999, Cramer et al., 1999).

A population-based case-control study performed in Pennsylvania, New Jersey and Delaware found a moderately increased risk of ovarian cancer with the use of talcum powder on feet, arms, and breasts, talcum powder in the genital region (OR 1.5, CI 1.1-2.0), as well as talcum powder use on sanitary napkins and underwear (Ness et al., 2000). No correlation was seen with duration of use of talcum powder. The risk for ovarian cancer was compared with 50 women with primary peritoneal cancers, and no control was established for confounders. Furthermore, the duration of use was based on the reported use of talc on the feet, genital, and rectal areas, but did not assess all the areas examined under the method of talcum powder use.

A cohort study in pulp and paper employees that showed an increased risk of ovarian cancer assessed potential risk factors associated with ovarian cancer, including the use of talc for personal hygiene (Langseth and Kjaerheim, 2004). The authors found no association between the hygienic use of talcum products and ovarian cancer.

A population-based case-control study of 22 counties in California found an increased risk of ovarian cancer in women who had ever used talc perineally (OR 1.37, CI 1.02-1.85) (Mills et al., 2004). While the authors found a trend of increasing risk with increasing frequency of talcum powder use, no such trend was found for duration of use or cumulative use of talcum powder.

The Nurses' Health Study is a cohort study that enrolled 121,000 registered nurses and assessed 78,630 participants' talc use and ovarian cancer diagnoses through 1996. The authors concluded that their results "provide little support for any substantial association between perineal talc use and ovarian cancer risk," finding no increase in risk of ovarian cancer with ever talc use (RR 1.09, CI 0.86 -1.37) and no increase in risk with increasing frequency of talc use. The authors did notice a modest increase with the risk of invasive serous ovarian cancer and talc use (RR 1.4, CI 1.02-1.91) (Gertig et al., 2000). The authors noted that the questionnaire used did not assess age of first talc use or the duration of use. A follow-up of the Nurses' Health Study and the Nurses' Health Study II in 2009 did not find a statistically significant association between ovarian cancer and talc use, although the authors noted a non-significant increased trend in risk for mucinous cancers with talc use greater than once a week. There was not an increase in risk with talc use for any other ovarian cancer subtype assessed (Gates et al., 2010).

In an Australian Ovarian Cancer Study, a population-based case-control study, the authors found a moderately increased risk of ovarian cancer associated with pelvic talc use (OR 1.17, CI 1.01-1.36) (Merritt et al., 2008). The authors note that there is no trend with duration of use, and that when examining subtypes, only serous tumors had a significant association with talc use. The authors included borderline malignant ovarian tumors in their assessment, and noted that cases were significantly older than controls, and had significant differences in parity, contraceptive use and previous reproductive surgery, all potential confounders.

In 2009, a population-based case-control study in Los Angeles county found a significant increase in the risk of ovarian cancer that increased with frequency and duration of talc use, one of the only studies to find an increase in risk with frequency and duration of talc use (Wu et al., 2009). The authors reported an increased risk with ever use of talc (RR 1.48, CI 1.15-1.91), including perineal use (RR 1.53, CI 1.13-2.09) and non-perineal use (RR 1.43, CI 1.03-1.98). The authors found no statistically significant increased risk with the use of talc on sanitary napkins, underwear or diaphragms. The risk increased with lifetime total times of talc use; however, this association was limited to individuals who began using talc before 1975. The authors attempted to examine the relationship between inflammation and the risk of ovarian cancer; interestingly, the authors also found an increased risk of ovarian cancer with increasing frequency and duration of anti-inflammatory use, in contrast to other studies; however, the authors did not differentiate between perineal use and any talc use in the assessment of frequency and duration. The authors failed to assess whether there are significant differences between cases and controls in regard to ethnicity and age. They also included both malignant and low malignancy ovarian disease in their study.

A population-based case-control study on white and African-American women in North Carolina assessed various risk factors and the different associations (Moorman et al., 2009). Significant differences in ovarian cancer stage and histological type were seen between the different ethnicities. The authors found no significant association with talc use and increased cancer risk in either group of women (OR 1.04, CI 0.82-1.33 and OR 1.19, CI 0.68-2.09). The authors note the small study size for African-American women, which limited evaluation of potential confounders, such as oral contraceptive use and tubal ligation.

A population-based case-control study found only a "*slightly increased,*" but not statistically significant risk of ovarian cancer (OR 1.27, CI 0.97-1.66) associated with perineal powder use, and noted no clear pattern on the basis of the extent of use, years used, or lifetime number of applications (Rosenblatt et al., 2011). The authors note that this increased trend is most evident among women diagnosed with borderline tumors (OR 1.55, CI 1.02-2.37). The authors found no increased risk with the use of talcum powder on sanitary napkins, diaphragms, or the use of vaginal deodorant spray. The authors concluded that "*no stronger adjective than 'possible' appears warranted at this time*."

A 2014 Women's Health Initiative Observational Cohort Study assessed the association between perineal powder usage and ovarian cancer (Houghton et al., 2014). The authors found that ever use of perineal powder (HR 1.06, CI 0.87-1.28) was not associated with ovarian cancer, nor was perineal powder application, the use of talc on sanitary napkins, or the use of talc on diaphragms. The authors note the potential for recall bias in case-control studies compared to their cohort study but noted that the potential for misclassification of exposure by participants was still present based upon the challenges of personal recollection of specifics regarding the use of perineal powder.

A 2015 analysis of ovarian cancer rates among women of different ethnic backgrounds examined non-genetic risk factors to see if these factors could explain the lower incidence of ovarian cancer in African-American and Hispanic women, respectively (Wu et al., 2015). Of interest, while African-American women had a 29% lower incidence of ovarian cancer, they had the highest percentage of talc use. The authors found across all ethnicities an increased association between ever talc use and ovarian cancer (OR 1.46, CI 1.27-1.69). The authors of this population-based case-control study did note that the risk associations with each of the risk factors assessed were comparable in the three groups but noted that talc use was not significantly associated with ovarian cancer in African American women. Additionally, among all three ethnic groups, the duration (length) of talc use did not correlate with ovarian cancer risk. The authors note a small sample size for Hispanic and African-American women, which did not allow for analyses by ovarian cancer type, and the authors only investigated six factors that they considered confirmed risk factors for ovarian cancer.

A population-based case-control study in New Hampshire and Massachusetts found an overall odds ratio between any genital talc use and ovarian cancer of 1.33 (CI 1.16-1.52) (Cramer et al., 2016). The authors adjusted this for age, study center and phase; however, they did not adjust this odds ratio for parity, contraceptive use, menarche, or other known confounders – this was done later in the study, where the authors found that these confounders had little to no effect on the genital talc use odds ratio. The authors found no association between body use of talc or the use of talcum powder on diaphragms, condoms, or partners. The authors did observe an increased risk with increasing frequency of use; however, no such

trend was found with the duration of use. The authors note a trend associated with estrogen, noting that association was "largely confined" to women who were premenopausal, or who were postmenopausal and had used hormonal therapy. The authors note the potential for misclassification, and that if the sensitivity of the study fell to 82% then the odds ratio for genital talc use and ovarian cancer would be null. The authors state that there is no way to assess records for talc use reported by study participants to see if this degree of misclassification is reasonable.

Gonzalez, et al. analyzed results of 41,654 participants from The Sister Study, a cohort study that enrolled and followed 50,884 women in the United States and Puerto Rico who had a sister diagnosed with breast cancer. The authors found that talc exposure was not associated with risk of ovarian cancer (HR 0.73, CI 0.44-1.2) (Gonzalez et al., 2016). Women who had previously been diagnosed with ovarian cancer or had their ovaries removed were excluded from the analysis, and the authors included women diagnosed with ovarian tumors, tumors of the fallopian tubes, peritoneum, or of uncertain origin (but likely from those three sites) as of July 2014.

Another population-based case-control study examining African-American women found that genital powder use was associated with an increased risk of ovarian cancer (OR 1.44, CI 1.11-1.86) and noted a dose-response relationship with the duration of use and number of lifetime applications (Schildkraut et al., 2016). Interestingly, this significance disappeared when the subject interviews were divided by those performed prior to 2014 and those performed after 2014. Genital and non-genital use of talcum powder was not associated with a significantly increased risk of ovarian cancer when interviewed prior to 2014 (OR 1.4, CI 0.96-2.03 and OR 1.19, CI 0.87-1.63) and genital use was only significantly associated with ovarian cancer risk when interviewed after 2014 (OR 2.91, CI 1.7-4.97). The authors note the potential for misclassification based upon self-reporting, "*especially due to heightened awareness of the exposure as a result of two recent class action lawsuits*." The authors further note the possibility for recall bias further inflating the odd ratios and note that the association with non-genital body powder use, which is not consistent with the available literature, indicates that misclassification of exposure and residual confounding cannot be ruled out.

7.3.2 Reviews and Meta-Analyses

A literature review published in 1994 (Wehner, 1994) stated that while talc has been identified in ovarian tissue, epidemiologic evidence associating talc use and ovarian cancer is generally weak and inconsistent, confounding variables are often ignored, reported increased risk ratios are often less than 2.0 (barely statistically significant), and epidemiological studies are not sensitive enough to estimate risk ratios less than 2.

A 2000 review of the literature examined perineal application of talc and cornstarch powders and noted that the available data indicated that "*associations between talc exposure and ovarian cancer have suggested but not proved a causal relationship"* (Whysner and Mohan, 2000).

A number of epidemiological reviews have assessed and listed potential risk factors for ovarian cancer, listing talc use as one of the potential factors (Hunn and Rodriguez, 2012, McLemore et al., 2009). These

reviews did not assess individual studies or perform any independent analysis of these studies and cited a subset of the studies discussed above.

A review in 2008 assessed the available scientific literature, noting that the heterogeneity in the perineal dusting studies and validity of exposure measurements, along with the lack of a consistent dose-response association, limits the ability to make causal relationship inferences (Muscat and Huncharek, 2008). The authors note that the use of cosmetic-grade talc at high doses for pleurodesis has not been associated with cancer in patients; nor have talc-dusted diaphragms and latex condoms. In performing a sensitivity analysis of perineal dusting studies, the authors found varying risks between hospital-based studies (OR 1.19, CI 0.99-1.41) and population-based studies (OR 1.38, CI 1.25-1.52). The authors noted potential variations based upon medical history and knowledge of risk factors, and further noted that while many of the studies included information on dose of talc exposure, few found a positive dose-response relationship. The authors published an additional study of scientific standards in epidemiology relating to the scientific approach regarding the potential causal association between talcum powder and ovarian cancer and conclude that the available epidemiological studies do not support a causal association (Huncharek and Muscat, 2011).

A 1995 meta-analysis of studies investigating talc exposure and ovarian cancer found the evidence to be inconclusive, with little to no evidence supporting a dose or duration relationship (Gross and Berg, 1995). The authors note that the relative risks in nearly every study with an association between talc exposure and ovarian cancer were below 2.0, and many contained biases and confounders that were not adjusted for as described above. In the ten studies examined by this meta-analysis, only four studies were primarily focused on talc exposure.

Similarly, a meta-analysis in 2003 found a relative risk of 1.33 (CI 1.16-1.45) when assessing 16 observational studies; however, the authors noted a lack of a clear dose-response relationship, "making the [relative risk values] of questionable validity" (Huncharek et al., 2003). The authors also noted that hospital-based studies showed no relationship between talc use and ovarian cancer risk (RR 1.19, CI 0.99-1.41) compared to population-based studies (RR 1.38, CI 1.25-1.52). The authors concluded that this suggests selection bias and confounding in population-based studies, and that the data do not support a causal association between perineal talc use and ovarian cancer.

A review in 2008 examined the association between talc use and ovarian cancer in one cohort study and 20 case-control studies (Langseth et al., 2008). The authors note that none of the studies they examined reported relative risks below 1.0. They further noted that the studies had varying degrees of significance, that four case-control studies and one cohort study provided results by ovarian cancer histological type, and that the cohort study in particular provided "hints of higher risks of serous tumors related to talc exposure." The authors stated that the main evidence against a causal association is the absence of a dose-response relationship in the majority of the studies and an absence of an excess risk in the cohort study examined.

A pooled study performed on eight population-based case-control studies found a "modest increased risk" of epithelial ovarian cancer (OR 1.24, CI 1.15-1.33) with genital powder use (Terry et al., 2013). In this analysis, the authors note there was no significant trend with increasing number of lifetime applications

of talcum powder and found no increased risk among women who reported non-genital talcum powder use. The authors noted that the prevalence of genital powder use in controls varied widely between the studies examined, and that significant differences between cases and controls included parity, tubal ligation, BMI, ethnicity, contraceptive use and talc use. The authors report varying trends with the type of ovarian cancer and note challenges in recall of talc products as a potential confounder, in addition to recall regarding dose and frequency of exposure.

A meta-analysis performed in 2018 to assess the risk of ovarian cancer from perineal talc use found 24 case-control studies and three cohort studies for examination (Berge et al., 2018). The authors found a relative risk for ovarian cancer of 1.22 (1.13-1.30), noting that this association was only found for serous carcinoma (RR-1.24, CI 1.15-1.34) and was limited to case-control studies with a suggestion of a dose response. The authors note that the "heterogeneity of results by study design however, detracts from a causal interpretation of this association." The authors also note that their estimate was lower than other meta-analyses, and that they "confirmed the trend toward lower overall risk estimate as more evidence accumulated." Another meta-analysis of 24 case-control studies and three cohort studies found that any perineal talc use was associated with a 24-39% increased risk of ovarian cancer (OR 1.31, CI 1.24-1.39) (Penninkilampi and Eslick, 2018).[3] Other statistics varied between case-control and cohort studies and types of ovarian cancer. The authors note that case-control studies were prone to recall bias, "especially with intense media attention following the commencement of litigation in 2014."

7.3.3 Studies on Talc, Inflammation, and Ovarian Cancer

Investigators have studied proposed mechanisms by which talc might initiate or contribute to a carcinogenic process, including whether talc causes inflammation or whether inflammation is itself related to the initiation of ovarian cancer. As discussed above in greater detail in part 6.4, inflammation has been suggested as a promoter of certain cancers due to its role in promoting cell growth. But in the context of the posited link between talc and ovarian cancer, the potential role of inflammation (if any) remains unsubstantiated. For example, in the Hamilton study discussed above in part 7.1, female rats were injected with talc, but no inflammation was observed. Moreover, scientific studies have shown that anti-inflammatory drugs do not reduce the risk of ovarian cancer (NCI, 2019) – a result that is difficult to square with the notion that ovarian cancer results from inflammation. Other studies, including the Merritt study discussed above in part 7.3.1, have found no statistically significant relationship between a history of pelvic inflammatory disease and any type of ovarian cancer, another result that is difficult to reconcile with the conception of ovarian cancer as a disease caused by inflammation. In short, the notion that either talc causes inflammation or that inflammation causes or promotes ovarian cancer remains unsupported.

7.3.4 Governmental and Agency Summaries

In 1995, a report was published following a 1994 workshop on talc sponsored by the FDA, the Cosmetics Toiletries, and Fragrances Association (CTFA), and the International Society of Regulatory Toxicology and

[3] I am advised that Dr. Eslick is an expert for the plaintiffs in this litigation; the source of funding for this analysis is not disclosed.

Pharmacology (ISRTP) (Carr, 1995). The panel concluded that with regards to ovarian cancer and talc exposure:

> "*…epidemiologic data are conflicting and remain equivocal. Although it is theoretically possible that talc could reach the ovaries, the actual access to or the presence of talc in ovarian tissue is not documented"* (Carr, 1995).

In an evaluation of the body of literature reviewed for talc not containing asbestiform fibers, the IARC working group determined that, despite some incongruencies in findings, there was limited evidence of an association between perineal application of talc and an increased risk for ovarian cancer. With regards to occupational exposure to talc, the IARC working group concluded that the data reviewed provided inadequate evidence to suggest an association between inhalational exposure to talc not containing asbestiform fibers and cancer in humans. (IARC, 2010).

In 2012, the Cosmetic Ingredient Review (CIR) Board convened an expert panel to assess the safety of talc used in cosmetic products (CIR, 2012). With respect to the potential association between the perineal use of cosmetic talc and ovarian cancer, the panel "*reviewed these studies thoroughly and determined that they do not support a causal link*" (Fiume et al., 2015, CIR, 2012). The panel noted that if the perineal applications of talc did increase the risk of ovarian cancer, then it would be expected that there would also be increased risk of uterine and cervical cancer, and that a similar increase in risk would be expected with the use of talc-dusted condoms or diaphragms.

In 2014, the FDA responded to two petitions requesting a cancer warning on cosmetic talc products. (Musser, 2014). In its response letter, the FDA denies the petition for a cancer warning, concluding that

> "*[The] FDA did not find that the data submitted presented conclusive evidence of a causal association between talc use in the perineal area and ovarian cancer"* (Musser, 2014).

A clinical commentary on the use of talc and association with ovarian cancer recommended not ascribing any particular case of ovarian cancer to talc use, due to the small effect size seen in the literature as well as a decreasing trend of talc use by women (Narod, 2016).

Most recently, in January of 2019 the National Cancer Institute (NCI) updated its Physician Data Query (PDQ) on ovarian cancer and addressed perineal talc use (NCI, 2019). The panel reviewed the available cohort studies and scientific literature and concluded that:

> *"the weight of evidence does not support an association between perineal talc exposure and an increased risk of ovarian cancer"* (NCI, 2019).

Conversely, Health Canada performed a meta-analysis and screening assessment of talc and concluded that the evidence indicated a "small but *consistent statistically significant positive association between ovarian cancer and perineal exposure to talc*" (Health Canada, 2018). The authors note that a clear point of departure has not been established, and thus any hazard characterization would be qualitative in nature.

7.3.5 Conclusion and Bradford Hill Criteria

In conclusion, the scientific literature regarding an association between talc use and ovarian cancer is varied and no consensus currently exists. Studies that have found an association between ovarian cancer and talc use have only been able to establish a weak association, with hazard ratios below 2, and the majority of studies have found no relationship between dose-response and increased risk. Additionally, the need for personal recall regarding talc use makes it hard to account accurately for the potential of recall bias and the potential misclassification of exposure.

The data are even more disparate and conflicting when "ovarian cancer" is disaggregated and the studies are examined by histological subtype. In 1999, for example, Cramer et al. reported varying odds ratios for different tumor types after adjusting for age and other covariates. In 2000, Gertig et al. showed an increased risk for serous invasive cancers, but not for an association between talc use and ovarian cancer risk more generally. In 2004, Mills et al. found an increased risk for serous invasive cancers specifically and an increased risk of epithelial ovarian cancer more generally, but not for certain other subtypes, such as serous borderline, mucinous (invasive or borderline), endometrioid or clear cell. In 2016, Cramer et al. found that risk varies by histologic subtype, among other factors. In short, these studies show no cross-study consistency between a certain type of ovarian cancer and talc exposure. While some show statistically significant associations with serous invasive ovarian cancer, others found no such association, or found an association with another subtype. (Harlow et al., 1992, Chang and Risch, 1997, Cook et al., 1997, Cramer, 1999, Wong et al., 1999, Gertig et al., 2000, Mills et al., 2004, Cramer et al., 1999, Merritt et al., 2008, Cramer et al., 2016).

When assessing this literature in the context of the Hill Criteria:

> *Strength* – The strength of the association between talc use and ovarian cancer is weak, with the studies that do show an association between the two only showing an increased risk of approximately 20-60%, respectively. This is much lower than the other confounders examined in these studies, such as hormone replacement therapy, oral contraceptive use, and parity. This is especially weak when compared to the example of cigarette smoking provided by Hill, which showed an increased risk of 20-30 times the general population (Hill, 1965).
>
> *Consistency* – As previously noted, there is little to no consistency among the scientific literature between these studies, as shown by the variation between population and hospital-based studies and between case-control and cohort studies, mode of genital talc application, and other places, circumstances and times.
>
> *Specificity* – As previously mentioned, the literature is varied about the types of ovarian cancer associated with talc use, with no general consensus on the type of ovarian cancer associated with talc use.
>
> *Temporality* – The literature regarding the temporality of exposure between talc use and ovarian cancer is also varied. No distinct latency period has been established between

talc exposure and the onset of ovarian cancer, and as previously mentioned, only a few studies found any relationship between length of exposure and risk of disease, or the age of exposure, if such information was available.

Dose-Response – The majority of studies found no association between the frequency, duration, or lifetime use of talc and increased risk of ovarian cancer.

Plausibility – The plausibility of a relationship between talc exposure and ovarian cancer relies solely on the proximity of talc particles to the ovaries, and the proposed migration to the ovaries. This is discussed in greater detail below and, as noted above, the lack of evidence of talc effect on the reproductive organs between the ovaries and genital area is an argument against this.

Coherence – The cause and effect hypothesis that talc exposure causes ovarian cancer generally relies on the potential migration pathway from perineal talc exposure and the induction of inflammation as being sufficient for carcinogenesis. As discussed throughout the various sections of this report, the generally known natural facts and biology regarding various aspects of talc, toxicology, ovarian cancer and exposure are not consistent with the assumptions necessary for a causal relationship between talc exposure and ovarian cancer.

Experimental Evidence – Several of these studies examined the potential for tubal ligation as a preventative measure to inhibit the transport of talc particles to the ovary, and thus prevent the alleged talc-based ovarian cancer. However, the limited associations already reported and the issues surrounding recall bias and misclassification, the significant association between ovarian cancer and other reproductive confounders, which would also be affected by tubal ligation, make this argument less than compelling.

Analogy – As previously described, judgment by analogy is the assessment of a similar compound or class of compounds when the current substance does not have sufficient research. Due to the amount of literature above specific to talc, an assessment of analogous components is not warranted or necessary. Furthermore, an analogous judgment using asbestos, as done by certain plaintiffs' experts, would be inappropriate, as discussed in Section 11.3, below.

As shown above, the Hill Criteria cannot be satisfied by the scientific literature, and thus a causal relationship between talc exposure and ovarian cancer is not the most likely relationship for the association shown in a subset of the described studies.

Response to Plaintiffs' Experts

Several of plaintiffs' experts in this litigation purport to apply the Hill causation criteria to varying degrees in support of their opinions that talc exposure causes ovarian cancer (including Drs. McTiernan, Kane, Clarke-Pearson, Siemiatycki, Smith, Wolf, Moorman, Smith-Bindman, Singh, and Carson). Not only does each expert apply different weight to different parts of the Hill Criteria, but for some of the parameters,

such as specificity and experimental evidence, they apply different interpretations. Several of these experts, when addressing the strength of the association, stress that while Hill originally stated that the greater the increased risk, the higher probability that a causative relationship exists, that is no longer the case and provide examples such as second-hand smoke and lung cancer, radon and lung cancer, and others with a more modest RR. However, the experts ignore the documented dose-response of these associations, choosing examples of lower exposure, which would result in lower relative risk compared to the higher relative risk associated doses that were integral to the identification and classification of these relationships. For example, direct smoking of cigarettes showed an increased lung cancer odds ratio as high as 103.5 or 111.3 (Pesch et al., 2012). No such dose-response has been established in the risk of talcum powder exposure and ovarian cancer, as discussed above, which would make a lower relative risk for lower level exposures more compelling.

Similarly, several of these experts state that the "majority" of the scientific literature shows an increased risk; however, as shown in the literature above, that is an inaccurate summary of the literature. Many of the studies that show an increased trend do not reach statistical significance (the confidence interval includes 1.0), or only one parameter reaches statistical significance, and the others do not. Additionally, several of plaintiffs' experts rely on the potential (yet unconfirmed) mechanism of exposure and toxicity as proof of causality, or the methodologically flawed expert reports of other experts such as Dr. Crowley and Dr. Longo regarding the presence of other materials, as discussed in this report.

Several experts also improperly use asbestos as an analogous substance for comparison, based upon their opinion or assumption that talc products contained asbestos (discussed further below), or upon analysis of industrial talc or talc ore.

8.0 Asbestos

Asbestos refers to a number of naturally occurring, fibrous minerals with high tensile strength, the ability to be woven, and resistance to heat and chemicals. Specifically, asbestos refers to the asbestiform versions of serpentine (chrysotile) and five amphibole (actinolite, amosite, anthophyllite, crocidolite, and tremolite) series minerals (IARC, 2012, Williams et al., 2007). These forms are defined by their unique physical and chemical characteristics set forth above. Due to these unique properties, asbestos fibers have been used in a wide range of manufactured goods, including insulation, ceiling and floor tiles, paper and cement products, textiles, coatings, friction products such as brakes and clutches, gaskets, and roofing shingles.

Because of the natural, commercial, and industrial occurrence of asbestos in the environment, everyone living in the United States is exposed to certain background levels of asbestos. Background exposure to asbestos can result from the presence of asbestos-containing materials (ACM) in urban and rural environments (HEI, 1991, Mangold, 1983, Nicholson et al., 1980, Churg and Warnock, 1980, Commins, 1989, Doll, 1987). Other exposures may occur in buildings that contain ACM (Ganor et al., 1992, Sawyer, 1977, USEPA, 1988b), near busy traffic areas of highways (Ganor et al., 1992), in the ambient air of urban cities; (WHO, 1998, Mangold, 1983, Corn, 1994), and in rural areas (HEI, 1991, WHO, 1998). Ambient exposures during the early 1980s ranged from 0.001 to 0.02 f/cc with exposures decreasing over time

(ATSDR, 2001, Mangold, 1983, Nicholson et al., 1980). This means the average American who lived in the 1980s is exposed to over 0.01-0.2 f/cc-years of asbestos over their lifetime attributable solely to this background level of ambient exposure.

8.1 Asbestos in Cosmetic Talcum Products

Beginning in the 1970s, several studies have analyzed potential exposure to asbestos fibers from the use of talcum products. In the peer-reviewed literature, five studies evaluate airborne dust concentrations associated with consumer use of talc products (Aylott et al., 1979, Russell et al., 1979, Moon et al., 2011, Gordon et al., 2014, Anderson et al., 2017).

A historical assessment in 1979 looked at the inhalational exposure to talc from the application of loose face powder, adult dusting powder and baby dusting power (Aylott et al., 1979). That same year, Russell and colleagues assessed the use and concentrations of talc in the air based on adults exposed to talc over their entire bodies and infants exposed in the napkin area (Russell et al., 1979). While these studies did not assess the presence of asbestos or fibers, they assessed the presence of respirable talc particles.

Another study in 2011 assessed the exposure risk through inhalation of baby powder for babies and adults (Moon et al., 2011). The authors conclude that the exposure concentrations were much lower than the threshold limit value for dust (TLV of 2 mg/m^3 set forth by the ACGIH). The authors further assessed the potential for lung asbestos contents and determined that any exposure resulting from the use of an asbestos-containing baby powder would be much lower than that of a normal individual with no asbestos-related occupational history. Two of the studies directly evaluate airborne asbestos exposures as a result of consumer use of talc products (Gordon et al., 2014, Anderson et al., 2017).

In 2014, Gordon and colleagues assessed 50 cosmetic talc products for the presence of asbestos. The authors examined 50 samples of one commercially available cosmetic talcum product based upon previous testing. (Gordon et al., 2014). The authors also performed two asbestos releasability studies, counting fibers according to the Asbestos Hazard Emergency Response Act (AHERA) protocols and Yamate et al. (1984), which consider particles with greater than 0.5 µm in length and at least a 5:1 aspect ratio a fiber. Nine samples were assessed via transmission electron microscopy (TEM) and found between 3 and 200 million asbestos fibers per gram that were greater than 0.5 µm in length with an aspect ratio of at least 5:1.

The results of the talc application tests are summarized in Table 8.1.1 below.

Table 8.1.1. Results from Gordon et al., 2014

Test	Amount Used	Phase Contrast Microscopy (PCM) Conc.	TEM Conc.
Shaker Application	0.37 g	4.8 f/cc (avg)	1.9 f/cc
Puff Application	6.25 g	20 f/cc (5 min)	4.25 f/cc

An exposure assessment from 2017 examined the potential inhalational exposure to talc particles and assessed for the presence of asbestos fibers in talc based upon historical cosmetic talcum powder use (Anderson et al., 2017). The author performed a simulated talc application study using historical talc samples believed to have been manufactured and sold in the 1960s and 1970s. Bulk analysis of the samples sent to two different laboratories showed conflicting results, with only one laboratory reporting the presence of asbestos fibers. The pertinent information and results are summarized in Table 8.1.2.

Table 8.1.2. Results from Anderson et al., 2017

Simulation Subject, Trial	Application Time per Event (seconds)	Total mass of Talcum Powder used* (g)	Respirable Dust (mg/m³)		Phase contrast microscopy (PCM) (f/cc)		Phase contrast microscopy equivalent (PCME) (f/cc)	
			Min	Max	Min	Max	Min	Max
5 Subjects, 2 Trials ea.	13-47	5.4-26.8	0.26	5.03	0.093	0.540	ND	0.0039**

* Total used over eight application events per trial (average 0.7-3.4 g per application)

** Classified as ambiguous, tentatively identified as anthophyllite, but further analysis could not be performed

In evaluating the data from all of these studies, the cumulative asbestos exposures arising from the use of talcum powder products fall far below the background level of asbestos that every person living in the United States experiences, as discussed above. The range of fiber concentrations during adult application of talcum powder in the three studies that examined fiber concentrations were from Not Detected – 5 f/cc, respectively, over a time frame ranging from 43 seconds up to five minutes (Anderson et al., 2017, Gordon et al., 2014, Dement et al., 1972). When extrapolated out to an eight-hour time weight average, this range becomes non-detect – 0.1 f/cc (assuming two applications of cosmetic talc per day). These levels would be of no causal significance for ovarian cancer as the IARC assessment of asbestos exposure and ovarian cancer found an association only with high levels of occupational exposure (IARC, 2012). For comparison, the asbestos exposure of asbestos textile workers historically ranged from 3-78 f/cc as estimated in the scientific literature (Dement et al., 2011, McDonald, 1998, Peto, 1980). These levels would be 30-780 times higher than the levels caused from adult application of talcum powder, assuming the highest alleged levels of exposure from the studies above. Drs. Longo and Rigler, two of plaintiffs' experts, have tested samples of talcum powder for asbestos, presenting data from which a worst-case cumulative dose exposure can be made. While for purposes of this evaluation, I will assume that all of the concentrations of asbestos presented by Drs. Longo and Rigler are accurate, I question the accuracy and relevance of the results.

The size and dimensions of asbestos fibers have significant implications in evaluating the potential effects on human health. For example, the WHO states that fibers with a diameter greater than 3 μm are not respirable and that fibers less than 5-10 μm in length are often engulfed by a single macrophage and cleared through the normal defense mechanisms of the body, but that fibers longer than this are more difficult to clear (WHO, 2000).

The Agency for Toxic Substances and Disease Registry (ATSDR) convened an expert panel to address the importance of fiber length in asbestos-related health effects. Based upon a review of human epidemiology and animal toxicology scientific literature, the panel concluded that asbestos and other fibers shorter than 5 µm are not likely to cause cancer (ATSDR, 2003).

A study in the 1980s found that short, large-diameter fibers were inactivated by phagocytosis, and that the probability of pleural sarcoma correlated best with fibers that were 0.25 µm or less in diameter and were more than 8 µm in length (Stanton et al., 1981).

In their report, Drs. Longo and Rigler note that they found bundles with diameters of 10-20 µm and lengths in excess of 50 µm. As discussed above, this would affect the aerodynamic capabilities of these structures and their ability to be inhaled into the lung or migrate within the body. Thus, the reporting of asbestos fibers of these sizes as a percentage of the overall weight of the sample examined instead of a numerical fiber concentration does not say anything about potential exposure to asbestos.

Despite this, in all the samples examined by Drs. Longo and Rigler, the amphibole asbestos reported as a weight percentage were <0.1%, even with the fiber counting metrics described above.[4] This by definition renders the products tested as non-asbestos-containing (United Nations, 2017). Even the AHERA TEM method mentioned by Dr. Longo and other experts defines an asbestos-containing material as a material containing ≥1% asbestos (USEPA, 1988a). According to this definition, anything with <1% asbestos fibers would not be considered asbestos-containing.

Furthermore, Drs. Longo and Rigler make the statement that consumer use of Johnson's Baby Powder and Shower to Shower "*would have, more likely than not, been exposed to significant airborne levels of both regulated amphibole asbestos and fibrous talc*" (Longo and Rigler Report, Page 32). Drs. Longo and Rigler provide no scientific basis for this statement, because (among other problems in their assessment) demonstrating asbestos content alone does not prove airborne exposure levels, as discussed above. Additionally, based on studies that have assessed airborne concentrations of asbestos fibers as a result of consumer use of talc, worst-case scenario exposures are still below even the most stringent occupational exposure limits, and are likely indistinguishable from background exposure based upon the majority of evidence.

Similarly, as discussed below, the Global Harmonization System (GHS) for the classification and labelling of chemicals, specifies that information is required for substances that are "*present above their cut-off/concentration limits*," which varies based upon the health-hazard criteria, with the most stringent cut-off being ≥0.1% (i.e., sensitizers, mutagens, or carcinogens) (OSHA, Undated). This is consistent with the FDA's 1985 risk assessment, in which the FDA assumed a 0.1% level of asbestos in talcum products and determined the exposure was below background (Brown, 1985). In this assessment, the FDA concluded that any hypothetical cancer risk due to asbestos fibers in talc was less than one in ten million, and possibly lower than this (Brown, 1985).

[4] It is only when using the Blount heavy liquid separation that Drs. Longo and Rigler found four samples with >0.1% weight asbestos fibers, and those were in the Asian historical samples that are not at issue in this litigation.

8.2 Asbestos and Ovarian Cancer

A 1960 case study in London found that women suffering from pulmonary asbestosis appeared to have ovarian neoplasms more often than the general population (Keal, 1960). A subsequent study in 1967 examined women diagnosed with ovarian cancer and performed animal studies in hamsters and mice. The authors found that intraperitoneal injection of asbestos caused surface abnormalities in the ovaries of the test animals and that the ovaries of women with ovarian cancer had histiocytes and foreign body giant cells about unidentified intact crystals, which were absent in normal ovaries (Graham and Graham, 1967). A 1972 study of female asbestos workers identified cases of ovarian cancer in former asbestos workers, although the number of cases was not large enough to draw definitive conclusions between asbestos exposure and ovarian cancer (Newhouse et al., 1972).

In 1982, a study of women who worked manufacturing gas masks from chrysotile or amphibole asbestos found an increased incidence of ovarian cancer in one of the two factories examined, although the authors voice concern about the diagnosis being confused with peritoneal mesothelioma (Acheson et al., 1982). A second study of female gas mask manufacturers was published in 1982. This study also found an increase in ovarian cancer, but similarly voiced concern about misdiagnosis of peritoneal mesothelioma, as follow up pathology reclassified one of three ovarian cases, with the remaining three cases unable to be assessed due to lack of sample (Wignall and Fox, 1982). Another cohort study in rock salt workers in Italy also found an increased incidence of ovarian cancer in female workers, despite the small study size (two ovarian cancers among 120 females) (Tarchi et al., 1994). A similar study on factory workers in London also found a statistically significant increased incidence of ovarian cancer in female workers that was higher with severe or lengthy exposure, though the study reported that the “total number of tumours remain[ed] low” (Newhouse et al., 1985).

A 1994 study of asbestos-exposed female workers in Germany found no increase in mortality rate to ovarian cancer than the general population (Rosler et al., 1994). One researcher who assessed talc burden of the ovaries also performed studies assessing asbestos burden of the ovaries of women with reported asbestos exposures. Asbestos was detected in four out of 12 women with reported exposure to asbestos and five out of 12 unexposed controls (Heller et al., 1996).

A study of women in the printing industry found an increased incidence of ovarian cancer in bookbinders, which the authors attributed to the use of asbestos-contaminated talc fillers in paper; however, no testing was done to verify asbestos exposure (Bulbulyan et al., 1999). A study of women in Italy from the textile and asbestos cement industries, selected based on their compensation for asbestosis, found increased risk of death due to ovarian cancer in both groups of workers (Textile Industry: SMR 526, CI 143-1347, Asbestos Cement Industry: SMR 540, CI 175-1261) (Germani et al., 1999). The authors state that these values were compared to the cause-specific mortality rates in the general population, although the method for this analysis is unclear.

A 2007 study investigated cancer mortality in a cohort of women married to asbestos workers in Casale Monferrato, Italy (Ferrante et al., 2007). Increased risk for deaths due to respiratory and pleural cancers was observed; however, there was no increased risk for ovarian cancer (SMR 1.42, CI 0.71-2.54). A 2009

study investigated ovarian and breast cancer risks in female workers exposed to blue asbestos in Australia (Reid et al., 2009). No consistent evidence for either ovarian or breast cancer was observed.

The IARC working group noted a causal association between heavy occupational exposure to asbestos and ovarian cancer (IARC, 2012). In response to this, a meta-analysis was performed that also concluded that heavy occupational exposure to asbestos is associated with increased risk of ovarian cancer (Camargo et al., 2011). Occupations examined included mining, textiles, gas mask assemblers, and mixed exposures (Camargo et al., 2011, Magnani et al., 2008). A cohort study of pulp and paper employees in Norway found no confirmed association between asbestos and ovarian cancer (Langseth and Kjaerheim, 2004). A literature review in 2011 found "*sufficient evidence to draw concern and warrant further investigation*" regarding the relationship between asbestos and ovarian cancer (Bunderson-Schelvan et al., 2011). A meta-analysis of the literature was performed in 2011, which reported that among women thought to have ovarian cancer, there was an increased rate among those who had reported asbestos exposure (Reid et al., 2011). The authors note that this result may have occurred due to disease misclassification, as the effect size dropped from 1.75 to 1.29 when only assessing studies that had pathologically confirmed cases of ovarian cancer. This study was included in a review article in 2017, which mentions that IARC declared that the evidence was sufficient to conclude that asbestos exposure can cause ovarian cancer despite the lack of consistency observed in the meta-analysis (Reid et al., 2017).

More recently, a study of Chinese asbestos workers found an increased incidence in ovarian cancer among female workers (Wang et al., 2013). One case of ovarian cancer was observed in the population of 277 female workers, when 0.13 cases were expected.

8.2.1 Conclusions

IARC's conclusions regarding the causal relationship between asbestos and ovarian cancer are a direct result of studies examining the heavy occupational exposure of women to asbestos products and fibers, not to consumer use of alleged asbestos-contaminated products, such as talcum powder. Some studies, such as the analysis of workers in Australia, did not show a link between asbestos and ovarian cancer; however, based on the scientific evidence, any potential link would only be associated with high levels of occupational exposure, as described above. As such, these exposure scenarios are drastically different from the types of exposures that would occur as a result of consumer use of talcum powder.

Plaintiffs' experts use the classification of asbestos as an ovarian carcinogen and opine that this, with the previously discussed opinions regarding asbestos content in talcum powder products, is evidence of a causal relationship between talcum powder exposure and ovarian cancer.

Even if asbestos fibers were present in cosmetic talcum powder products, then the studies and analyses discussed in Section 7.3, which failed to establish a causal relationship between talc use and ovarian cancer, would also include asbestos fibers.

9.0 Heavy Metals

9.1 Chromium

Chromium is a naturally occurring element in the earth's crust that is found in both natural and man-made sources (ATSDR, 2012b). According to the ATSDR, the industries with the largest consumption and release of chromium include metal processing, tanneries, chromate production, welding of stainless steel and chrome pigment production (ATSDR, 2012b).

People may be exposed to chromium by inhaling ambient air, eating chromium-containing food, or drinking chromium-containing water. Dermal exposure to the general population may occur through contact with certain consumer products and chromium-contaminated soils. For the general public, the primary method of exposure is food ingestion (ATSDR, 2012b). The primary method of occupational exposure to chromium is through the inhalation of vapors and/or particulate-containing hexavalent chromium.

The toxicokinetics and behavior of chromium depend on the oxidation state of the chromium atom, which refers to the number of valence electrons gained or lost on the chromium atom. Chromium (III) is the most stable oxidation state. Chromium (III) is an essential nutrient for glucose, fat, and protein metabolism, and is frequently found in oral supplements (ATSDR, 2012b). Chromium (III) has also been used in the chemical industry in pigments and in leather tanning. Chromium (III) is considered relatively non-toxic for humans and other mammalian species (IPCS, 1988). Studies have shown that while Chromium (III) is toxic to intracellular constituents, the ion is not transported across the cell membrane (Norseth, 1986). There is currently insufficient evidence that Chromium (III) is carcinogenic in humans or experimental animals (IARC, EPA). The carcinogenicity classifications of Chromium (III) are included in Table 9.1.1, below.

Table 9.1.1: Chromium (III) Carcinogenicity Classification

Agency	Classification
IARC	Group 3 (Not classifiable as to its carcinogenicity to humans)
NTP	NA
USEPA IRIS	D – Not classifiable as to human carcinogenicity

Chromium (VI), also called hexavalent chromium, is the second-most-stable form of chromium; however, it rarely occurs naturally (USEPA, 1984). Chromium (VI) compounds exist in a variety of chemical structures of varying solubility and are primarily generated during manufacturing processes. Chromium (VI) compounds have been classified by IARC as Group 1 (*Carcinogenic to humans*) classification for lung cancer (IARC, 2018). The USEPA has determined chromium (VI) to be carcinogenic to humans through inhalation based on their chromium (VI) integrated risk information system (IRIS) assessment (USEPA, 2018a). Additionally, the NTP has determined that chromium (VI) compounds are known to be human carcinogens based on sufficient evidence from studies in humans, which is documented in the fourteenth

edition of their RoC. The carcinogenicity classifications of Chromium (VI) are included in Table 9.1.2, below.

Table 9.1.2: Chromium (VI) Carcinogenicity Classification

Agency	Classification
IARC	Group 1 (Known Human Carcinogen)
NTP	Known to be Human Carcinogens
USEPA IRIS	A – Carcinogenic to Humans

Literature searches yielded no mechanistic or epidemiologic studies linking chromium exposure to ovarian cancer.

9.2 Nickel

Nickel is a hard, silvery-white metal that is naturally occurring in the earth's crust, with a long history of industrial use. The chemical properties of nickel make it useful for forming alloys, including nickel alloy and stainless steel. Nickel salts are used in electroplating and as a component of nickel-cadmium batteries. Additionally, nickel sulfide is used as a catalyst in chemical reactions.

Nickel compounds have an IARC Group 1 (Known Human Carcinogen) classification for nasal cavity/paranasal sinus and lung cancer (IARC, 2018). The USEPA has determined nickel compounds, nickel refinery dust, and nickel subsulfide to be carcinogenic to humans based on their respective IRIS assessments (USEPA, 2018a). Additionally, the NTP has determined that nickel compounds are known to be human carcinogens based on sufficient evidence from studies in humans, which is documented in the fourteenth edition of their RoC. The carcinogenicity classifications of nickel and nickel compounds are included in Table 9.2.1, below.

Table 9.2.1: Nickel and Nickel Compounds Carcinogenicity Classification

Agency	Classification (Nickel and Nickel Compounds)
IARC	Group 1 (Known Human Carcinogen)
NTP	Known to be Human Carcinogens
USEPA IRIS	A – Carcinogenic to Humans

There is currently insufficient evidence in the scientific literature to associate nickel exposure and ovarian cancer. To date, a single study was found, which assessed nickel accumulation in ovary epithelial cells in patients with diagnosed epithelial ovarian cancer (Canaz et al., 2017). Nickel concentrations were found to be higher in ovarian tumor tissue than normal, healthy ovary tissue. However, it cannot be determined from the author's data if nickel exposure contributed to the development of ovarian cancer or if accumulation of nickel within the tissue is a consequence of the development of ovarian cancer. Thus, no causal relationship can be determined. Additionally, no nickel exposure data, patient health information, or other risk factors for ovarian cancer are assessed by the authors of the study.

9.3 Cobalt

Cobalt and cobalt-containing compounds exist in many forms, each with their own uses in industrial applications. The chemical and physical properties of cobalt make it useful for forming cemented carbides and alloys, particularly superalloys used in aerospace engineering and superalloys with magnetic properties. Cobalt also has historically been used to create blue dyes for paint and pottery.

Exposure to cobalt in the general population occurs through inhalation of ambient air and ingestion of food and drinking water. Human activities that deposit cobalt into the environment include burning of fossil fuels, applications of cobalt-containing sludge and phosphate fertilizers, mining and smelting of cobalt, processing of cobalt-containing alloys, and industries that manufacture or use cobalt-containing compounds (ATSDR, 2004). Cobalt is a component of vitamin B12 and is essential to the human body. The Recommended Dietary Allowance of vitamin B12 is 2.4 µg/day, which contains 0.1 µg of cobalt by weight (ATSDR, 2004).

Regulatory agencies have reviewed the toxicity of cobalt in its metallic form, cobalt-containing alloys, and cobalt-containing chemical compounds. Cobalt metals with tungsten carbide have an IARC Group 2A classification (Probably Carcinogenic to Humans) for lung cancer. Cobalt metals without tungsten carbide, cobalt sulfate, and other soluble cobalt (II) salts all have an IARC Group 2B (Possibly Carcinogenic to Humans) classification for nasal cavity/paranasal sinus and lung cancer (IARC, 2018). The USEPA has not yet performed an IRIS assessment of cobalt, so it currently remains unclassified. The NTP evaluates cobalt carcinogenicity in two categories: cobalt and cobalt compounds that release ions in living tissue and powder and metals containing cobalt-tungsten carbide. The carcinogenicity classifications of cobalt-tungsten carbide and other cobalt compounds are included in Table 9.3.1, below.

Table 9.3.1: Cobalt-Tungsten Carbide and Cobalt Compounds Carcinogenicity Classification

Agency	Classification	
	Cobalt-Tungsten Carbide	**Other Cobalt Compounds**
IARC	Group 2A classification (Probably Carcinogenic to Humans)	Group 2B (Possibly Carcinogenic to Humans)
NTP	Reasonably Anticipated to be a Human Carcinogen	Reasonably Anticipated to be a Human Carcinogen
USEPA IRIS	NA	NA

Literature searches yielded no mechanistic or epidemiologic studies linking cobalt exposure to ovarian cancer.

9.4 Lead

The predominant use of lead in most countries is as a metal that can be alloyed with other materials. However, the largest use of lead worldwide is in the production of lead-acid batteries. Lead has been phased out of many products, such as pipes, paint, and gasoline, in favor of other options.

Inorganic lead compounds have an IARC Group 2A (Probably Carcinogenic to Humans) classification, while organic lead compounds have an IARC Group 3 (Not Classifiable as to their Carcinogenicity to Humans) classification (IARC, 2018). The IARC review included occupational exposure studies that investigated lung, stomach, and kidney cancers, as well as brain and nervous system cancers. The USEPA has determined inorganic lead compounds to be probable human carcinogens based on animal studies reviewed in their IRIS assessment that reported increases in renal tumors (USEPA, 2018a). Additionally, the NTP classified lead compounds as reasonably anticipated to be human carcinogens based on limited evidence of carcinogenesis in multiple tissue sites from studies in humans and sufficient evidence from studies showing kidney, brain, and lung tumor development in experimental animals, which is documented in the fourteenth edition of their RoC. The carcinogenicity classifications of inorganic lead are included in Table 9.4.1, below.

Table 9.4.1: Inorganic Lead Carcinogenicity Classification

Agency	Classification
IARC	Group 2A (Probably Carcinogenic to Humans)
NTP	Reasonably Anticipated to be a Human Carcinogen
USEPA IRIS	B – Probably Carcinogenic to Humans

There is currently insufficient evidence in the scientific literature to associate lead exposure and ovarian cancer. To date, a single study was found, which assessed lead accumulation in ovary epithelial cells in patients with diagnosed epithelial ovarian cancer, which is the same study above that also investigated nickel accumulation (Canaz et al., 2017). Lead concentrations were found to be higher in ovarian tumor tissue than normal, healthy ovary tissue. The same study deficiencies that were identified for nickel are also true for lead. It cannot be determined from the authors' data if lead exposure contributed to the development of ovarian cancer or if accumulation of lead within the tissue is a consequence of the development of ovarian cancer, and thus no causal inference can be made. Additionally, no lead exposure data, patient health information, or other risk factors for ovarian cancer are assessed by the authors of the study.

9.5 Cadmium

Cadmium has many properties that make it desirable for industrial use, including corrosion resistance, low melting temperature, and high thermal and electrical conductivity. The predominant use of cadmium is in the production of nickel-cadmium batteries, with some other uses in pigmentation, coating and plating, and as a stabilizer for plastics.

In the United States, the primary sources of exposure to cadmium in the public are from food ingestion and tobacco smoke. Tobacco leaves naturally accumulate high amounts of cadmium from soil, which results in an estimated 1.7 µg of cadmium per cigarette. About 10% of the total cadmium in a cigarette is inhaled when smoked (ATSDR, 2012a). Leafy vegetables such as lettuce and spinach can contain relatively high levels of cadmium.

Cadmium and cadmium compounds have an IARC Group 1 (Carcinogenic to Humans) classification for lung cancer (IARC, 2018). The USEPA has determined cadmium to be a probable human carcinogen based on limited human studies showing evidence of increased lung cancer and animal studies reporting lung cancer development from inhalation that were reviewed in their IRIS assessment (USEPA, 2018a). Additionally, the NTP classifies cadmium and cadmium compounds as known to be human carcinogens based on sufficient evidence of carcinogenesis from studies in humans that reported increased risks of lung cancer, which is documented in the fourteenth edition of their RoC. The carcinogenicity classifications of cadmium are included in Table 9.5.1, below.

Table 9.5.1: Cadmium Carcinogenicity Classification

Agency	Classification
IARC	Group 1 (Carcinogenic to Humans)
NTP	Known to be Human Carcinogens
USEPA IRIS	B – Probably Carcinogenic to Humans

Literature searches yielded no mechanistic or epidemiologic studies linking cadmium exposure to ovarian cancer in humans. However, three studies were found showing no association between ovarian cancer rates and dietary cadmium intake (Adams et al., 2014, Eriksen et al., 2014, Julin et al., 2011).

9.6 Arsenic

Arsenic is a naturally occurring element than can be found in both soils and minerals. In industry, arsenic has been used as a pesticide/herbicide in agriculture, and arsenic compounds have been used as anti-fungal wood preservatives, pharmaceuticals, components in metal alloys, and in the manufacture of semiconductors and other specialized electronics. As of 2007, approximately 90% of arsenic produced was used as a wood preservative to prevent rot (ATSDR, 2007a).

The general population is exposed to arsenic through ingestion of food and drinking water, as well as inhalation from breathing ambient air. Crops grown in areas with high soil arsenic levels, either from natural sources or historic use of arsenic as a pesticide, will contain higher levels of arsenic than crops grown elsewhere (ATSDR, 2007a). Additionally, groundwater typically contains higher levels of arsenic than surface water, as the geology forming water tables beneath the earth's surface tends to have higher concentrations of arsenic than surface soils (ATSDR, 2007a).

Inorganic arsenic compounds have an IARC Group 1 (Carcinogenic to Humans) classification for lung, bladder and skin cancer (IARC, 2018). The USEPA has determined inorganic arsenic to be a human carcinogen based on human studies reporting increases in lung, internal organ (liver, kidney and bladder) and skin cancers that were reviewed in their IRIS assessment (USEPA, 2018a). Additionally, the NTP classified arsenic and inorganic arsenic compounds as known to be human carcinogens based on sufficient evidence of carcinogenesis from studies in humans, which is documented in the fourteenth edition of their RoC. The carcinogenicity classifications of arsenic are included in Table 9.6.1, below.

Table 9.6.1: Arsenic Carcinogenicity Classification

Agency	Classification
IARC	Group 1 (Carcinogenic to Humans)
NTP	Known to be human carcinogens
USEPA IRIS	A – Human carcinogen

Literature searches yielded no mechanistic or epidemiologic studies linking arsenic exposure to ovarian cancer in humans. Studies on mice from the lab of Michael Waalkes showed that exposure to very high levels of arsenic exposure in drinking water during gestation can induce ovarian tumors in offspring (42.5 ppm for 2 weeks of gestation or 6 ppm during gestation with continued exposure over the lifetime of the offspring) (Waalkes et al., 2004, Tokar et al., 2011, Waalkes et al., 2007, Waalkes et al., 2003). Additionally, there are studies investigating the effectiveness of inorganic arsenic as a potential treatment for ovarian cancers (Luo et al., 2018, Zhang and Wang, 2006, Zhang et al., 2013, Chun et al., 2002).

9.7 Non-Occupational Exposure to Heavy Metals in Daily Life

Heavy metals are ubiquitous in the environment and humans are exposed to them daily during routine activities, such as eating, drinking and breathing. Multiple studies have been performed to understand the amount of metals consumed by the average individual each day. One of these, the Total Diet Study, is an ongoing FDA program that monitors the levels of approximately 800 food contaminants and nutrients in the average US diet. The FDA carries out this program by purchasing and analyzing approximately 280 types of foods and beverages from different areas of the country four times per year (USFDA, 2018).

Table 9.7.1. FDA Total Diet Study – Average Daily Consumption (µg/day)

Age	Nickel[a]		Cobalt[a]		Total Arsenic[b]	
6-11 Months	68.8		3.4		2.15	
2 Years	90.4		5.2		23.4	
	Male	**Female**	**Male**	**Female**	**Male**	**Female**
14-16 Years	162.5	118.9	11.6	7.6	15.4	21.8
25-30 Years	146.2	106	10.8	7.2	56.6	27.5
60-65 Years	130.8	99.6	9	6.3	92.1	72.1

[a] Pennington and Jones (1987)
[b] Tao and Bolger (1998)

Of the six metals cited by the plaintiffs, nickel has the highest daily consumption in the United States. Analysis from the FDA Total Diet Study estimates an average daily intake between 68.8 and 162.5 µg/day depending on age and gender (Table 9.7.1), while the ATSDR has previously estimated an average daily intake of between 69-162 µg/day for adults (Table 9.7.2). While nickel concentrations in food vary by type of foodstuff and the region in which it was produced, the highest concentrations of nickel in foods have been measured in beans, seeds, nuts and grains.

The primary source of non-occupational exposure to cobalt comes from dietary intake in food and water. As a component of vitamin B12, consumption of cobalt is also essential for human health. Analysis from

the FDA Total Diet Study estimates an average daily intake between 3.4 and 11.6 µg/day depending on age and gender (Table 9.7.1). The ATSDR estimates the average person consumes roughly 11 µg of cobalt daily in their diet (Table 9.7.3). Cobalt levels in surface and ground water typically range between 1-10 µg/L in populated areas and are lower in rural areas (Hamilton, 1994; Smith and Carson, 1981 as cited in ATSDR, 2004). Foods that contribute most heavily to cobalt intake are baked goods, cereal and vegetables.

The primary source of non-occupational exposure to arsenic comes from dietary intake in food and water. Arsenic exists in many chemical forms and several oxidation states, and analysis from the FDA Total Diet Study estimates an average daily intake between 2.15 and 92.1 µg/day depending on age and gender (Table 9.7.1). However, only a fraction of total arsenic intake would be in the inorganic form. The ATSDR estimates total dietary inorganic arsenic in the United States to be 1-20 µg/day (Table 9.7.3). The primary source of inorganic arsenic in US diets are from grains and produce.

The greatest sources of lead exposure arise from its previous use as an additive in paint and gasoline, the latter of which resulted in widespread dispersal across the United States roadways and surrounding areas. Dispersion from these historical sources makes estimating daily exposure to lead difficult, as it is highly context dependent. The NTP estimated the average daily intake of dietary lead in 1990 to be roughly two-nine µg depending on age (Table 9.7.2). In contrast, the ATSDR estimates one µg lead/kg body weight consumed in the diet daily, which results in 70 µg/day lead consumption for a 70 kg adult.

Table 9.7.2. NTP Report on Carcinogens, 14th Edition – Estimate of Daily Lead Intake in 1990 (µg/day)

Age	Lead
2 Years	4
14-16 Years	6-9
25-30 Years	6-9
60-65 Years	2-8

The primary source of non-occupational exposure to chromium comes from consumption of chromium-containing foods. As previously discussed, chromium (III) is an essential nutrient required for energy metabolism as determined by the Institute of Medicine of the National Research Council. The ATSDR estimates the mean dietary chromium intake of the general US population is 0.505 µg/kg body weight per day, or 35.35 µg/day for the average 70 kg adult (Table 9.7.3). The highest concentrations of chromium can be found in meat, fish, fruits and vegetables (ATSDR, 2012b).

The primary source of non-occupational exposure to cadmium also comes from dietary intake. The ATSDR estimated the mean dietary cadmium intake of the general US population is 0.32 µg/kg body weight per day, or 22.4 µg/day for the average 70 kg adult (Table 9.7.3). Foods known to contain high amounts of cadmium include leafy vegetables like lettuce and spinach, as well as potatoes and grains. Additionally, shellfish and organ meat from animals can contribute to cadmium exposure for individuals who consume them regularly (ATSDR, 2012b).

Table 9.7.3. ATSDR Estimates of Daily Intakes in Adults (µg/day)

Metal	Daily Intake
Chromium (Food)	35.35[a]
Nickel (Water)	4-8.6
Nickel (Food)	69-162
Lead (Food)	70[a]
Cadmium (Food and Air)	22.4[a]
Cobalt (Food)	11[a]
Inorganic Arsenic (Food)	1-20

[a] Calculated using ATSDR document daily intake per kg body weight, assuming an average adult body weight of 70kg.
Table adapted from ATSDR (ATSDR, 2012b, ATSDR, 2005, ATSDR, 2007b, ATSDR, 2012a, ATSDR, 2007a, ATSDR, 2004).

9.8 Heavy Metals in Talc

There are limited studies assessing the concentration of heavy metals in talc and talc-containing products. In the 1968 study previously discussed (Cralley et al., 1968), the authors examined the presence of metals in 22 commercial talcum products. While the authors did not elucidate what methodology was performed, they stated that with the exception of three products, the products had concentrations of metals below the following:

- Cobalt – <25 ppm
- Chromium – <22 ppm
- Nickel – <29 ppm
- Manganese – <78 ppm

While many heavy metals have been identified as human carcinogens, it is notable that none of the heavy metal contaminants of talc identified by the Cralley study have an IARC Group 1 (Known Human Carcinogen) classification for ovarian cancer. While Chromium (VI) and nickel compounds have an IARC Group 1 classification, the classification is for cancers other than ovarian. Cobalt in various forms is listed in Groups 2A (cobalt with tungsten carbide, associated with lung cancer), 2B and 3, and manganese is unlisted.

The expert reports of Dr. Mark Krekeler and Dr. Robert Cook refer to testing in various talc products provided by IMERYS and the Johnson & Johnson defendants in their assessment of the metal components of talcum powder, specifically in regard to the talcum products mentioned above. These documents are provided in detail in Appendix C and are summarized below in Table 9.8.

Table 9.8. Summary of IMERYS and Johnson & Johnson Defendants' Heavy Metals Documents

Product Tested	Analytes Examined/Reported (mg/kg)*					
	Chromium	Nickel	Cobalt	Lead	Cadmium	Arsenic
Baby Powder	ND – 400	30-2,050	0.0-66	0.2-0.5		0.4-<3
Other Samples	0.25-8,500 (<4-70 ppb CrVI)	<0.2-2,650	0.1-96	0.05-41	<0.02-3	<0.2-2,100

* As cited by Dr. Cook and Dr. Krekeler. All results have been converted to mg/kg (ppm = mg/kg).

As shown, the range of detections of the aforementioned heavy metals is large, spanning more than an order of magnitude in most cases. Samples included baby powder, talc, talc or historical talc drill samples, mine sludge, grade 66 talc, and other species. Additionally, talc samples came from multiple locations and areas and were analyzed by varying methods, making comparison for informative purposes difficult. In the case of the crude talc ore, which had some of the highest detections for heavy metals, this is not comparable to the final product, which is processed via magnetic separation and acid washing to remove minerals and metals (Fiume et al., 2015). The levels of these metals in baby powder samples were well below screening values established by the USEPA for dermal exposure to particulates/soils containing these metals (USEPA, 2018b).

9.9 Conclusions

In summary, none of the heavy metals listed by plaintiffs' experts has been causally associated with ovarian cancer. Plaintiffs' experts opine that the classification of these heavy metals as probable, possible, or known human carcinogens for the various forms of cancer above can be extrapolated to assume their carcinogenicity to the ovary as well. However, as specified in the Hill Criteria as discussed above, specificity is one of the criteria by which causal relationships are determined; and the extrapolation to other forms of cancer is not appropriate.

Additionally, as described above, the majority of these metals are needed for the normal and healthy function of the human body. The general population is exposed to all these metals on a regular basis through food, water, air and other sources. While there are not any screening values for products such as talcum powder, the USEPA has derived screening values for materials such as tap water and soil/particulates and dermal exposure, establishing risk-based criteria determined to be protective of human health as previously described in Section 6.2. All of the concentrations discussed in Table 9.8 above are well below these values.

None of plaintiffs' experts examines the role of dose in the assessment of heavy metals in talcum powder, nor does any plaintiffs' expert go further than a cursory association between ovarian cancer and any other form of cancer. As shown above, the scientific literature does not support an association between any of these metals and ovarian cancer, and the concentrations present in baby powder are below screening levels established for dermal exposures. As such, there is no scientific basis for the opinion that heavy metals contribute to talcum powder's alleged causal relationship with ovarian cancer.

10.0 Fragrant Chemicals in Talc

10.1 Potential carcinogens

10.1.1 Styrene

Styrene is a compound used in the manufacture of plastics, latex paints and coatings, synthetic rubbers, polyesters, and styrene-containing coatings. It is present in indoor and outdoor air and can be found in food as a result of styrene compounds used in food packaging materials. Styrene is also found in cigarette smoke. Human exposure to styrene can occur both occupationally and non-occupationally. Occupational exposure to styrene accounts for much higher levels of exposure to styrene than non-occupational exposure and occurs primarily in styrene manufacturing and processing, reinforced glass production, and the manufacture of styrene-butadiene rubbers. For non-occupational exposure, the primary sources of styrene exposure are cigarette smoke, air inhalation and food intake. The daily intake of styrene for the non-smoking general population has been estimated at 0.3-0.8 µg/kg bodyweight per day, 90% of which results from air inhalation (IARC, 1994). The carcinogenicity classifications of styrene are included in Table 10.1.1, below.

Table 10.1.1: Styrene Carcinogenicity Classification

Agency	Classification
IARC	2B – possibly carcinogenic to humans*
NTP	Reasonably anticipated to be a human carcinogen
USEPA IRIS	Not evaluated

*Styrene is currently classified as group 2A (probably carcinogenic to humans) by IARC (IARC, 2018); however, the final report for this classification is currently in preparation at the time of this report. Styrene was previously classified as group 2B by IARC in 2002.

The carcinogenicity classifications of styrene in humans are primarily based on occupational exposure studies that show the potential for increased mortality from or incidence of lymphohematopoietic cancer, which are cancers relating to the production of lymphocytes and blood cells and include cancers of the blood, bone marrow, spleen, lymph nodes or thymus (Bond et al., 1992, Delzell et al., 2001, Delzell et al., 1996, Hodgson and Jones, 1985, Sathiakumar et al., 1998, Sielken and Valdez-Flores, 2001, NTP, 2008). Some studies reported increased risk for esophageal and pancreatic cancer in workers exposed to styrene; however, no causal relationship was established due to the potential for confounding by other substances (Hodgson and Jones, 1985, Kolstad et al., 1995, NTP, 2008).

Literature searches yielded no mechanistic or epidemiologic studies linking styrene exposure to ovarian cancer in humans.

10.1.2 Para-cresol (4-methylphenol)

Para-cresol (p-cresol, also known as 4-methylphenol) is one of three forms of cresols, which are compounds that are used as food additives, flavoring agents, solvents, disinfectants, deodorizers, and chemical intermediaries. The primary routes of exposure to p-cresol are inhalation and oral ingestion

associated with the use of consumer products that contain cresols. p-Cresol (4-methylphenol) has not been evaluated for carcinogenic potential by IARC or NTP. The carcinogenicity classifications of p-Cresol (4-methylphenol) are included in Table 10.1.2, below.

Table 10.1.2: P-Cresol (4-Methylphenol) Carcinogenicity Classification

Agency	Classification
IARC	Not evaluated
NTP	Not evaluated
USEPA IRIS	C – possible human carcinogen

The USEPA classifies p-cresol as a possible human carcinogen based on increased incidence of skin-papillomas in mice (USEPA, 1991). The only available human carcinogenicity data is anecdotal. One study reported two cases of multifocal transitional cell carcinoma of the bladder following chronic occupational exposure to cresol and creosote. An additional study reported one case of vocal cord cancer in a petroleum worker with a history of exposure to cresol, dichlorooctane, and chromic acid (USEPA, 1991). Literature searches yielded no mechanistic or epidemiologic studies linking para-cresol exposure to ovarian cancer.

Literature searches yielded no mechanistic or epidemiologic studies linking para-cresol exposure to ovarian cancer in humans.

10.1.3 Coumarin

Coumarin is widely distributed in the plant kingdom and has a significant history of commercial use. It is primarily used as raw material for fragrances in soaps, detergents, lotions, perfumes and other cosmetic products. Occupational exposure to coumarin occurs primarily in the production of coumarin and the manufacture and processing of cosmetics that use coumarin. Non-occupational intake of coumarin has been estimated at 0.06 mg/kg bw and is comprised of both dietary intake and exposure from use of cosmetics. Coumarin has been shown to exhibit immune system modification and direct antitumor activity and has been recommended for treatment of a number of clinical conditions. At the time of its assessment by IARC, coumarin was undergoing clinical trials for treatment of tissue swelling following breast cancer treatment, lung and kidney cancer and melanoma (IARC, 2000). Coumarin has not been evaluated for carcinogenic potential by NTP or USEPA. The carcinogenicity classifications of coumarin are included in Table 10.1.3, below.

Table 10.1.3: Coumarin Carcinogenicity Classification

Agency	Classification
IARC	3 – not classifiable as to its carcinogenicity to humans
NTP	Not evaluated
USEPA IRIS	Not evaluated

There are no carcinogenicity studies available on coumarin exposure in humans; as such, IARC's classification for coumarin is based on experimental animal studies. An oral administration study of mice exposed to coumarin showed significantly increased incidence of lung tumors at the highest dose in both males and females and increased incidence of liver tumors in females at the low and medium doses (NTP, 1993 as cited in IARC, 2000). A second oral administration study of mice exposed to coumarin showed no significant increase in the incidence of tumors outside the historical control range for the laboratory (Carlton et al., 1996 as cited in IARC, 2000).

Literature searches yielded no mechanistic or epidemiologic studies linking coumarin exposure to ovarian cancer.

10.1.4 Eugenol

Eugenol is primarily used as a fragrance and flavoring agent, numbing agent, an insect attractant, and a chemical intermediate. It is the principal compound in oil of cloves, which is used as a raw material in fragrances. It has been identified in many types of commercial alcohols, particularly those aged in barrels such as whiskies, cognacs, brandies and rums. Primary exposure to eugenol is via the ingestion of consumer products that contain eugenol. IARC reported that daily intake of eugenol was estimated at 0.605 mg/day (IARC, 1985). Eugenol has not been evaluated by carcinogenicity by NTP or USEPA. The carcinogenicity classifications of eugenol are included in Table 10.1.4, below.

Table 10.1.4: Eugenol Carcinogenicity Classification

Agency	Classification
IARC	3 – not classifiable as to its carcinogenicity to humans
NTP	Not evaluated
USEPA IRIS	Not evaluated

There are no carcinogenicity studies available on eugenol exposure in humans; as such, IARC's carcinogen classification for eugenol is based on experimental animal studies. An oral administration study of eugenol exposure in mice by NTP showed a statistically significant increase in instances of kidney tumors in female mice. The same study found no significant increase in incidence of tumors in rats orally exposed to eugenol (NTP, 1983 as cited in IARC, 1985). There was no statistically significant increase in instances of kidney tumors in male mice, and there was no increase of tumors at other sites compared to controls. An oral exposure study of mice by Miller et al. (1983) showed no increase in the instance of tumors, though IARC noted that the Miller experiments are of short duration (as cited in IARC, 1985).

Literature searches yielded no mechanistic or epidemiologic studies linking eugenol exposure to ovarian cancer.

10.1.5 d-Limonene

d-Limonene is one of the most common terpenes found in nature. It is naturally occurring in citrus and other plant species and is a major constituent of citrus rind, dill oil, oil of cumin, neroli, bergamot and

caraway. It is used as a flavor and fragrance additive to food, beverages, and consumer products; a solvent; and in the manufacture of resins. Occupational exposure to d-limonene primarily occurs during its manufacturing and usage as an industrial solvent. Non-occupational exposure to d-limonene can occur from food, drink, consumer products, and indoor and outdoor air, with an estimated daily intake of 0.3 mg/kg bw (IARC, 1999). At the time of its most recent assessment by IARC, d-limonene was undergoing trials for usage in the treatment of breast cancer and other tumors. d-Limonene has not been evaluated for carcinogenicity by NTP or USEPA. The carcinogenicity classifications of d-limonene are included in Table 10.1.5, below.

Table 10.1.5: d-Limonene Carcinogenicity Classification

Agency	Classification
IARC	3 – not classifiable as to its carcinogenicity to humans
NTP	Not evaluated
USEPA IRIS	Not evaluated

There are no carcinogenicity studies available on d-limonene exposure in humans; as such, IARC's carcinogen classification for d-limonene is based on experimental animal studies. In its original assessment of d-limonene in 1993, IARC cited two oral administrations studies of d-limonene, which showed no treatment-related tumors in mice or rats. In its second assessment of d-limonene in 1999, IARC included four additional experimental animal studies of d-limonene, three of which showed decreases in the incidence of cancers in subjects treated with d-limonene. In an oral administration and intraperitoneal injection study of mice, El-Bayoumy et al. (1996) found a decrease in the number of lung tumors in mice treated with d-limonene (as cited in IARC, 1999). A study by Kawamori et al. (1996) similarly found that mice treated with d-limonene had decreased frequencies of colon cancer and numbers of tumor per colon, and a study of pancreatic cancer development by Nakaizumi et al. (1997) found significantly decreased incidences of pancreatic tumors in hamsters treated with a high dosage of d-limonene (as cited in IARC, 1999). In a multi-organ model of cancer development, Kimura et al. (1996) found no incidences of renal tumors in rats treated solely with d-limonene.

Literature searches yielded no mechanistic or epidemiologic studies linking d-limonene exposure to ovarian cancer.

10.2 Irritants, Sensitizers and Allergens

The talc product ingredients added to the products at issue for purposes of fragrance are evaluated for their potential as irritants, sensitizers or allergens. I begin by reviewing the criteria described in the GHS for hazard classification for classifying compounds, mixtures, or substances as irritants, sensitizers or allergens. The GHS is provided on the OSHA website in conjunction with the OSHA Hazard Communication Standard (HCS). Data from the peer-reviewed scientific literature, the National Library of Medicine's TOXNET database, RTECS database, U.S. FDA, and the CIR compendium were also consulted for information.

The seventh revised edition of the GHS guidance (United Nations, 2017) describes the hierarchy of hazard classification for skin irritation and sensitization and describes an approach for considering the weight-of-evidence for the available data to make hazard classification decisions. Section 3.2 of the GHS guidance defines skin irritation as "…the production of reversible damage to the skin occurring after exposure to a substance or mixture." Section 3.4 defines skin sensitization as "…an allergic response occurring after skin contact with a substance or a mixture." GHS guidance provides for two tiers of irritation or sensitization hazard classification, depending on the results seen in human or laboratory animal data. Specific clinical endpoints and effect scores are described in the GHS guidance for skin irritation effects. Likewise, effects criteria for human clinical reports, epidemiology studies and animal immunology assays are described in the guidance for classifying sensitization hazards. The GHS guidance recommends higher weight be given to data collected using standardized toxicity testing methods, such as the Organization for Economic Co-operation and Development (OECD) Guidelines for the Testing of Chemicals. These criteria are considered for skin irritation and sensitization hazard classification for the following six talc product fragrance compounds.

The concentration of ingredients of a mixture dictate whether the mixture itself is classified as a skin irritant or as hazardous to the skin. Samples of these mixture guidelines are summarized in the tables below.

Table 10.2.1. Tables A.2.3 and A.2.4 from GHS Guidelines for the Testing of Chemicals – Regarding Classification of a Mixture as Hazardous to the Skin

	Concentration triggering classification of a mixture as	
Sum of Ingredients Classified as:	**Skin Corrosive – Category 1**	**Skin Irritant – Category 2**
Skin Category 1	≥ 5%	≥ 1% but < 5%
Skin Category 2		≥ 10%
When additivity does not apply:		
Ingredient:	**Concentration:**	**Classified as skin:**
Acid with pH ≤ 2	≥ 1%	Category 1
Base with pH ≥ 11.5	≥ 1%	Category 1
Other corrosive (Category 1) ingredients	≥ 1%	Category 1
Other irritant (Category 2)	≥ 3%	Category 2

Similar concentration triggers are listed in the GHS regarding eye irritation and sensitizers. It is important to note that the GHS notes a difference between respiratory and dermal sensitizers, with concentration triggers of ≥ 0.1% (OSHA, Undated).

It is also important to note that based upon the GHS guidelines for irritation and sensitization, none of the fragrant chemicals that make up the fragrance used in the products at issue would cause the product to be classified as a corrosive, irritant, or sensitizer, regardless of their individual classifications as irritants, corrosives or sensitizers. Appendix D shows a detailed list of the fragrant components of Johnson's Baby Powder, as well as their concentrations in the product itself, compared with a subset of available toxicity information from the above sources. A subset of these fragrant chemicals are discussed in more detail

below, with specific reference to studies regarding irritation, corrosion and sensitization and the doses associated with those responses.

10.2.1 Amyl cinnamal (CAS No. 122-40-7)

Amyl cinnamal is also known as alpha-amyl cinnamaldehyde. It is used as a fragrance and flavoring and has been used extensively in soap products (HSDB, 2018). Some example concentrations of amyl cinnamal in various food products are shown in Table 10.2.1 (HSDB, 2018).

Table 10.2.1. Reported Uses of Amyl Cinnamal

Food Category	Usual (ppm)	Max (ppm)
Alcohol beverages	0.47	1.22
Baked goods	6.44	9.69
Frozen dairy	2.67	5.19
Gelatins, puddings	4.19	6.78
Hard Candy	9.84	9.84
Nonalcoholic beverages	2.61	4.47
Soft Candy	5.55	7.65

The Hazardous Substances Database (HSDB) states that sensitivity to amyl cinnamal is uncommon, but can accompany a perfume allergy, and it is a skin irritant (HSDB, 2018). Studies were not provided that showed the concentrations at which skin irritation occurred.

10.2.2 Carum carvi (caraway) fruit oil (CAS No. 8000-42-8)

Caraway oil is a natural product extracted from the dried Carum Carvi fruit. It is used as a flavor ingredient in foods and has been used in traditional medicine due to its antimicrobial properties, beyond its use in the perfume and soap industry as a fragrance agent (HSDB, 2018). Example concentrations of Caraway oil in various food products are shown in Table 10.2.2.

Table 10.2.2. Reported Uses of Caraway Oil

Food Category	Usual (ppm)	Max (ppm)
Alcohol beverages	114.8	142.4
Baked goods	181.6	225.4
Cheese	0.05	0.12
Chewing Gum	0.31	0.45
Condiments	36.03	41.71
Frozen Dairy	34.95	43.43
Nonalcoholic beverages	27.11	35.09*
Meat Products	110.9	146.0

Several studies were performed in human subjects to assess potential sensitization and irritation at concentration of 4% and 1%, none of which showed an adverse reaction in the volunteers (HSDB, 2018).

10.2.3 Geraniol (CAS No. 106-24-1)

Geraniol is an oily liquid that has a rose-like odor and a citrus taste. Geraniol occurs naturally in more than 200 plants, including tea, grapes, apricots and plums (HSDB, 2018). It has been used in perfumes and food flavorings, as well as in natural tick and animal repellants. Example concentrations of geraniol in various food products are shown in Table 10.2.3 (HSDB, 2018).

Table 10.2.3. Reported Uses of Geraniol

Food Category	Usual (ppm)	Max (ppm)
Alcohol beverages	0.93	2.76
Baked goods	17.0	25.08
Chewing Gum	33.02	43.15
Frozen Dairy	8.58	12.00
Gelatins, Puddings	2.11	4.85
Gravies	1.5	3.00
Nonalcoholic beverages	3.49	5.86
Soft Candy	15.92	24.44

A human patch test found that geraniol at concentrations of 32% was severely irritating, and a second exposure study found that geraniol in 2% concentrations caused contact (dermal) allergy (HSDB, 2018). Another study performed a patch test and found that 10.5-25% of individuals exposed to geraniol at 6.0% and 11.0% react to the geraniol (HSDB, 2018).

10.2.4 Linalyl acetate (CAS No. 115-95-7)

Linalyl acetate has a floral/fruity odor and sweet taste and is found in the oils of many plants. It is frequently used as a flavor additive and in perfumes and paints. The HSDB notes that linalyl acetate is "generally recognized as safe" (GRAS) for human consumption (HSDB, 2018). Example concentrations of linalyl acetate in various food products are shown in Table 10.2.4 .

Table 10.2.4. Reported Uses of Linalyl Acetate

Food Category	Usual (ppm)	Max (ppm)
Alcohol beverages	0.55	1.55
Baked goods	15.67	23.34
Chewing Gum	1.91	25.82
Condiments	5.00	40.00
Frozen Dairy	8.67	12.41
Gelatins, Puddings	11.5	18.07
Nonalcoholic beverages	5.53	8.46
Hard Candy	4.04	23.43
Soft Candy	9.19	15.28

The HSDB notes that linalyl acetate is a "severe skin irritant" (HSDB, 2018). A concentration of 100% linalyl acetate applied to rabbit skin was defined as severely irritating and defined as moderately irritating when applied to guinea pig skin. A patch test of 100% linalyl acetate on miniature swine caused no irritation (HSDB, 2018). In human subjects, a 20% linalyl acetate solution produced no irritation, nor did a 2% solution. A study in humans with dermatitis found that a 6.0% linalyl acetate solution produced a contact allergy in 2.2% of the population examined (HSDB, 2018).

10.3 Conclusions

In conclusion, none of the fragrant chemicals noted by plaintiffs' experts has been associated with ovarian cancer. As shown in Appendix D, the fragrant chemical components make up an extremely low percentage of the talcum powder products at issue, a fact that is ignored by Dr. Crowley and the other plaintiffs' experts that rely on his assessment. None of these chemicals is present in the talcum powder products at issue at levels that would warrant a health or regulatory concern, as discussed below.

Furthermore, Dr. Crowley and other plaintiffs' experts' opinions regarding irritation, sensitization and inflammation in associating these fragrant chemicals with ovarian cancer are inherently flawed. As discussed above, dose is intimately tied with irritation and an inflammatory response, and thus to disregard the dose required for irritation, sensitization or inflammation and extrapolate that the induction of inflammation is sufficient to establish a causative relationship with ovarian cancer is not only overly simplistic, but a misrepresentation of the mechanisms of carcinogenicity (as described in Section 6.4), and basic toxicological principles (as described in Section 6.1).

11.0 Additional Methodological Flaws in the Expert Report of Dr. Plunkett

11.1 Dr. Plunkett's use of studies involving talcs other than cosmetic talc is based on the faulty assumption that since cosmetic talc powders products are not 100% pure platy talc, all studies on talcs of varying purity levels are relevant, without further context.

Dr. Plunkett's assertion that *"women using talcum powder products… were exposed to a mixture of chemicals, not 100% pure platy talc"* and thus, *"studies that describe talc products of varying purity levels were relevant to the assessment,"* ignores critical distinctions between cosmetic talc and industrial talc (Plunkett Rep., paragraph 29). Dr. Plunkett's reliance on literature regarding exposures to various forms of talc, including tremolite talc, mining talc, and industrial talc, is misguided (Plunkett Rep., paragraphs 38 and 39). These constitute very different talcs from cosmetic-grade talc; yet, Dr. Plunkett ignores these differences and merely assumes the relevance of studies analyzing these talcs to her assessment. That assumption is without basis for several reasons.

First, cosmetic and other talcs may contain varying levels of potential impurities, but Dr. Plunkett made no attempt to compare the relative percentage of potential impurities in cosmetic talc to the talc analyzed in the cited studies. Dr. Plunkett did not determine whether any of the talcs analyzed in those studies ever ended up in the products at issue (Plunkett Deposition, pages 135-36). Dr. Plunkett was not willing to opine, however, that industrial-grade talc that might contain up to 70% tremolite presents the same level

of toxic effect as cosmetic talc that may contain no (or very little) tremolite (Plunkett Deposition, page 145). Without a comparison of the relative percentage of potential impurities in cosmetic talc and the talc in those studies, there is no basis to conclude that those studies provide relevant evidence regarding the alleged toxic effect of cosmetic talc.

Second, many of the studies cited by Dr. Plunkett involved occupational exposure settings. But as Dr. Plunkett conceded, an occupational setting would result in exposures much higher than consumer exposures to a cosmetic talc product (Plunkett Deposition, pages 174-75). Dr. Plunkett did not conduct any analysis comparing exposure levels in an occupational setting to the anticipated exposure level for a consumer using cosmetic talc (Plunkett Deposition, pages 176-77).

Third, Dr. Plunkett notes that the diseases associated with occupational exposures to various forms of talc are lung cancer and fibrotic changes (talcosis and pneumoconiosis), not ovarian cancer or mesothelioma (an asbestos-related disease) (Plunkett Report, paragraph 39).

11.2 Dr. Plunkett regularly ignores references to dose, as well as the importance of dose in toxicology and in the assessment of exposures, health risks, and disease.

As previously discussed, and as acknowledged by Dr. Plunkett, dose is the key hallmark of the study of toxicology (Plunkett Deposition, pages 293-94 ("fundamental principle")). Any compound on earth, whether natural or man-made, medicinal or necessary for life, can be "toxic" if the dose and/or route of exposure are appropriate. Dr. Plunkett's opinion that Dr. Hildick-Smith's paper "*list[ed] many studies that provide proof that talc has toxic properties at certain doses and by different routes of exposure. i.e., talc itself is a toxic compound*," entirely ignores dose (Plunkett Report, paragraph 39).

Dr. Plunkett's statement that talc is a toxic compound has no meaning without additional context regarding dose, exposure pathways, and endpoints such as ovarian cancer, fibrosis, etc. Dr. Plunkett even states in Paragraph 40 of her report that "*[t]he types of [talc] toxicity produced are dependent on the route of exposure and the purity of the talc product,*" although she fails to mention dose.

In paragraph 68 of her report, Dr. Plunkett again ignores dose in stating, "*[w]hen considered together with general principles of toxicology, the available data relating to mechanism of carcinogenicity of talcum powder products… indicate that the various compounds in talcum powder products would be expected to produce at least an additive effect on the risk of cancer*…"

Dr. Plunkett again ignores the important role of dose in these conclusions and erroneously concludes that there would be an additive effect simply because a portion of the components of talcum powder, ***at a sufficient dose***, may be carcinogenic. Dr. Plunkett's conclusion is pure speculation. To understand potential additive effects, a study of compound endpoints (target organs, symptoms, etc.) would have to be undertaken as well as the mechanism of action of the compound and the dose-response. Compound mixtures may have additive effects, synergistic effects, no effect, or inhibitory effects on one another, depending on these criteria. Neither Dr. Plunkett nor anyone else to her knowledge has conducted such a study (Plunkett Deposition, pages 223-25, 292-93).

Dr. Plunkett's disregard for the importance of dose is particularly suspect given her opinion in paragraph 69 of her report that talc "*may be a non-genotoxic carcinogen,*" which "*requires repeated dosing of sufficient duration for tumors to be produced*." As previously discussed in sections 6.3 and 6.4, the role of a threshold dose has been shown to apply not only to mutagens, but to promoters and co-carcinogens, and non-genotoxic carcinogens.

Dr. Plunkett opines that *"[talc] exhibits a threshold for tumor development, produced tumors that exhibit a dose-response relationship with exposure…and may exhibit species, strain, and tissue specificity*" (Plunkett Report, paragraph 69). As a result, Dr. Plunkett testified that there would be a threshold dose below which talc would not have a carcinogenic effect (Plunkett Deposition, pages 281-82). Dr. Plunkett, however, cannot identify—nor point to any scientific literature identifying—that threshold (Plunkett Deposition, page 282). Moreover, neither Dr. Plunkett nor anyone else has established that long-term perineal application of cosmetic talc would result in a sufficient delivery of dose to the ovaries to cross that unidentified threshold.

11.3 Dr. Plunkett mistakenly opines that fibrous talc would have similar toxicity and should be treated the same as asbestos and relies on Dr. Longo's reports and inaccurate measurements and fiber counting to opine regarding the contamination of talc products with asbestos.

Dr. Plunkett notes in paragraph 31 of her report that *"[i]f a fiber is long, immune cells cannot totally engulf the compound and remove the foreign materials from the tissue,"* concluding that any potential adverse effects from fibers would be similar, whether asbestos or talc in chemical composition. Dr. Plunkett contradicts this in her deposition when she states that it is not her opinion that fibrous talc has the same toxic potential as tremolite (Plunkett Deposition, page 151).

As previously discussed, not only does fiber size plays an important role in fiber toxicity, but many other factors do as well, including biopersistence and surface chemistry, shown by the varying toxicities of different types of asbestos (i.e., chrysotile versus amphibole asbestos) (Hodgson and Darnton, 2010, Hodgson and Darnton, 2000). This is further elucidated by toxicological assessments performed by IARC and the ATSDR, which discuss variations between the biopersistence and toxicity of asbestos fibers, but do not include fibrous talc as having similar toxicity (IARC, 2012, ATSDR, 2001). A study in 1981 assessed the various toxicities of fibrous minerals, including amphibole asbestos and fibrous talc (Stanton et al., 1981). The authors found that size played a key role in toxicity, as discussed in Section 8.0, but also that talc showed a less than expected response compared to other fibrous minerals, especially tremolite, which the authors noted had a close affinity to the talcs.

Dr. Plunkett also refers to Dr. Longo's assessment of asbestos in talcum powder and the detection of asbestos in cosmetic talcum products (Plunkett Report, paragraphs 32 and 33). Dr. Plunkett applies no assessment of the methodology used to ascertain the presence of asbestos. Furthermore, Dr. Plunkett stated in her deposition that her opinions would not change if the product did not contain asbestos (Plunkett Deposition, pages 257-59).

11.4 Dr. Plunkett's opinions regarding the presence and hazards related to fragrant chemicals in the talc products at issue not only ignore dose and exposure, but also the scientific literature regarding these compounds.

In discussing the fragrance added to the talcum powder products at issue in paragraph 34 of her report, Dr. Plunkett erroneously states that *"the 2017 document produced by Johnson & Johnson… fail[s] to provide specific information on the amount of each chemical component in the fragrance component"* in the product.

The 2017 document that I have been provided by counsel for the Johnson & Johnson defendants (from their document production in this litigation) does indeed provide a range of the percentage of each compound in the fragrance component. This information, taken together with the formula declaration report, provides the amount of each chemical component of the fragrance in Johnson's Baby Powder (i.e., 0.22% of the total weight). This information is examined further in the Response to Dr. Crowley below and Appendix D.

Dr. Plunkett does not assess the dose-response, symptoms or endpoints of the compounds that she states "*have been associated with potential carcinogenic activity*" or have been linked with some level of irritation (Plunkett Report, paragraph 35). Dr. Plunkett does not examine the scientific basis for the carcinogenic ratings, nor the type of cancer associated with the compounds, as previously discussed in Section 10.1. Dr. Plunkett acknowledged that merely because a compound can cause one type of cancer does not mean that it can cause all types of cancer (Plunkett Deposition, page 269). For example, Dr. Plunkett testified that chromium is classified by IARC as a known human carcinogen for some gastrointestinal and skin cancers yet conceded that she had not conducted any analysis evaluating whether it would be appropriate to extrapolate that classification to ovarian cancer (Plunkett Deposition, pages 271-72). Dr. Plunkett further conceded that she has not conducted any analysis; nor could she identify any scientific studies establishing that any of the heavy metals or fragrance compounds that she cites as potential constituents of the products at issue increase the risk for ovarian cancer (Plunkett Deposition, pages 274-75). As previously discussed in this report (Sections 9.0 and 10.0) none of the potential carcinogens in fragrant chemicals or heavy metals that Dr. Plunkett refers to are associated with ovarian cancer.

Dr. Plunkett also states that the components of the fragrance used in Johnson's Baby Powder have been linked with "*some level of irritant hazard to tissues,"* and makes the general statement that irritation is related to carcinogenesis (Plunkett Report, paragraph 35), but again, she does not examine or take into account the doses or mechanisms of these compounds. As discussed earlier in section 6.4, causing irritation does not mean a compound will induce cancer, and irritation is a general symptom that is highly dose-related.

11.5 Dr. Plunkett's theory of particle migration from the genital area to the ovary has not been established in the scientific literature and is severely flawed.

Dr. Plunkett's migration theory, whereby talc particles supposedly migrate through the body – either through perineal application or inhalation – and arrive at foreign tissues, such as the ovaries, is severely flawed (Plunkett Report, paragraphs 41-56).

First, Dr. Plunkett's migration theory for perineal application would require talc to migrate upwards – against gravity and the downward flow of bodily fluids in the female reproductive tract – through the uterus, cervix and fallopian tubes; however, no pathology is associated with talc exposure and these portions of the female anatomy. Indeed, IARC has concluded that "*the evidence for retrograde transport of talc to the ovaries in normal women is weak*" and animal studies have "*shown[n] no evidence of retrograde transport of talc to the ovaries*" (IARC, 2010). If Dr. Plunkett's migration theory were correct, one would expect an increase in uterine, cervical and vaginal cancers, but none has been reported. To the extent Dr. Plunkett is still proffering an inhalation theory (contrary to her deposition testimony (Plunkett Deposition, page 176), that theory is flawed for the same reason: If talcum powder reached the ovaries through inhalation, one would expect an increase in lung cancer or mesothelioma, but Dr. Plunkett does not identify any studies evidencing such an increase.

Second, Dr. Plunkett concedes that many of the studies she cites regarding reproductive migration are not based upon talc particles, but other "*inert particles*" (Plunkett Deposition, pages 74, 183). Dr. Plunkett acknowledges that "*only if a particle is one that is similar to talc*" would its ability to move up the reproductive tract in women be relevant (Plunkett Deposition, page 70). Dr. Plunkett has not, however, analyzed whether the particles in those studies are similar to talc. For example, she relies on a study regarding the migration of cornstarch, but undertook no analysis regarding any differences between the migration patterns of cornstarch and talc (Plunkett Deposition, pages 69-71).

Third, Dr. Plunkett's observation that talc particles have been found in the ovaries of women who reported using talcum powder products on the genital area does not demonstrate causation. Dr. Plunkett ignores that talc has also been observed in the ovaries of women who have never used talc. For example, in the Heller study cited by Dr. Plunkett as support, the researchers found that "*talc particles were observed to a similar extent with both exposed and unexposed subjects*"(Heller et al., 1996). The presence of talc particles in ovarian tissue plainly is not sufficient to indicate migration and/or that the compound is responsible for the disease.

Fourth, Dr. Plunkett makes the erroneous connection that the ability of a particle to be moved through the female reproductive tract must causally link the particle to ovarian cancer. The notion that talc travels to the ovaries and causes cancer via inflammation fails to account for the fact that studies have rejected an association between use of talc-dusted diaphragms and condoms and ovarian cancer" (Fiume et al., 2015, Huncharek et al., 2007).

11.6 Dr. Plunkett's risk assessment does not establish or opine as to causation and does not meet the methodology of a true risk assessment as described in Section 6.2.

In her deposition, Dr. Plunkett stated that she did not perform a general causation analysis in her report, but rather performed a risk assessment to determine whether a hazard relevant to human health exists. Dr. Plunkett further testified that it is not her intent to offer the opinion that Johnson's Baby Powder *causes* ovarian cancer, but that it is her opinion that it "*increases the risk of ovarian cancer*" (Plunkett Deposition, pages 34-35). As previously discussed in Section 6.2, risk assessment cannot be used to establish causation. Indeed, risk assessment cannot even be used to predict actual health effects that hazardous substances at a site may have on people, because rather than provide an accurate evaluation of true health risk, risk assessment is intended to be precautionary and health protective. Without a causal relationship, Dr. Plunkett is merely assuming or opining as to a correlation between the use of talcum powder products and ovarian cancer. Dr. Plunkett goes so far as to state in her deposition that she does not agree that the strength of evidence has to be greater in order to determine if an agent causes a disease compared to determining if an agent increases risk (Plunkett Deposition, pages 38-39). Not only is this contrary to the principles underlying any causality assessment, including the Hill Criteria, but it further ignores the role of causation in a true risk assessment. On page 157 of her deposition, Dr. Plunkett states that she did not quantify a cancer potency factor, but that she is quantifying whether or not she believes the risk of ovarian cancer is increased. This is pure speculation, as the lack of a quantifiable cancer potency factor means that a quantifiable risk assessment is impossible (USEPA, 1989). Interestingly, later in Dr. Plunkett's deposition, she states that she cannot quantify the risk of the fragrant components of the talcum powder products without knowing the concentrations of these products, in direct contradiction of her previous opinions (Plunkett Deposition, pages 168-70).

Moreover, the methodology applied by Dr. Plunkett in conducting her "risk assessment" is flawed. As discussed earlier, the framework for risk assessments has been researched and developed by multiple agencies for the assessment of risk and the protection of human health, and it is these guidelines that must be followed when performing a risk assessment to ensure that an accurate, appropriate, and health-protective assessment is performed. Rather than following the established framework, Dr. Plunkett applied her own methodology, whereby she purported to use a weight-of-the-evidence approach but applied no quantitative values to particular pieces of evidence (Plunkett Deposition, pages 85-89). Instead, Dr. Plunkett testified that her methodology was simply the use of her experience, training and judgment to gather and evaluate information on exposure and response (Plunkett Deposition, pages 77-78). When discussing other health and screening assessments, such as Health Canada or other organizations, Dr. Plunkett testified that those organizations use their own methodology for risk assessment, whereas she uses her methodology (Plunkett Deposition, pages 202-04). Dr. Plunkett's application of "*experience, training and judgmen*t" in an unrecorded and non-replicable manner is no methodology at all.

11.7 Conclusion

For the reasons stated above, Dr. Plunkett's methodology for assessing the potential toxicity of Johnson's Baby Powder and its components and her risk assessment regarding whether a hazard relevant to human

health exists is flawed and not based upon sound science, the backing of regulatory bodies, or acceptable industry practice.

12.0 Additional Methodological Flaws in the Expert Report of Dr. Zelikoff

12.1 Dr. Zelikoff's opinion that talcum powder contains "*known carcinogens*" and compounds that "*elicit an inflammatory response*," the presence of which provides evidence of a "*causal relationship between genital use of talc and ovarian cancer*" ignores endpoints and dose (Zelikoff Report, page 12).

Dr. Zelikoff makes reference to the carcinogenicity of various alleged components – including heavy metals and fragrances – of the talcum powder products at issue, but entirely fails to examine the basis for the carcinogenic ratings or the forms of cancer associated with these compounds. As discussed above, there is no basis to conclude that a compound that can cause one type of cancer can cause all types of cancer. But, with respect to the heavy metals at issue, Dr. Zelikoff conceded that none of the studies she relies on specifically addressed ovarian cancer (Zelikoff Deposition, pages 280-82). Dr. Zelikoff likewise did not identify any literature analyzing the fragrances at issue and ovarian cancer. Dr. Zelikoff merely states that she reviewed the expert report of Dr. Crowley and concurs with his opinions, rather than performing any independent assessment of her own (see Section 15.0). Furthermore, Dr. Zelikoff did not perform a causation analysis at all, but rather stated in her deposition that she was asked to assess the "biological plausibility" of talc products causing ovarian cancer (Zelikoff Deposition, page 156).

Dr. Zelikoff's opinion also fails to consider the dose of the alleged "*known carcinogens*" and "*inflammatory agents*." It is worth reiterating that all substances can be toxic at certain exposure levels, as previously described in this report. A dose level is a key component in evaluating whether an exposure will result in an adverse health event. Dr. Zelikoff herself testified that "*it's important to look at dose-response relationships*" in analyzing the toxicity of an agent (Zelikoff Deposition, page 343). Yet, Dr. Zelikoff neither analyzed – nor identified any studies establishing – the dose at which talcum powder, or any of the substances she contends it contains, would begin the biologic process leading to ovarian cancer. Indeed, Dr. Zelikoff stated that dose is "*unknown*" and not reported in the scientific literature (Zelikoff Deposition, pages 265-66).

Dr. Zelikoff also erroneously surmises that a single exposure of an unknown concentration "*can produce effects" and that* "*it can start the process of either inflammation or oxidative stress."* (Zelikoff Deposition, page 369). As discussed earlier in this report (Section 6.0), and as stated by Dr. Zelikoff herself, the dose makes the poison, a tenet that her assertion that a single exposure can be causative ignores. As discussed, the concentrations of heavy metals, fragrant chemicals, and other allegedly carcinogenic substances, are insufficient to reach a threshold for any adverse health effect, including irritation, and furthermore, in the case of heavy metals and fragrant chemicals, none has been implicated or associated with ovarian cancer in the scientific literature.

Dr. Zelikoff is reduced to the unsupportable assertion that talc and its components are capable of causing inflammation that could lead to cancer at any dose. Dr. Zelikoff concedes that no published authority

supports that opinion (Zelikoff Deposition, pages 440-41). As previously discussed, promoters of inflammation in carcinogenesis require sustained, repeated exposures and exhibit a dose threshold, and by themselves do not cause cancer (Section 6.4). Furthermore, as discussed above in part 7.3.3, there is no evidence that inflammation is a cause of ovarian cancer; in fact, for example, scientific studies have shown that anti-inflammatory drugs do not reduce the risk of ovarian cancer (NCI, 2019).

Dr. Zelikoff takes this faulty reasoning one step further by concluding that other agencies such as NTP, IARC, and the Institute of Medicine have concluded that there is biologic plausibility between perineal talc use and ovarian cancer because they stated that "*talc… produces inflammation*" (Zelikoff Deposition, pages 222-23). These agencies do assess biologic plausibility in their assessments; however, as discussed in the Hill Criteria in Section 5.0, this is but one factor of a causal analysis that is performed by each agency.

12.2 Dr. Zelikoff's opinions that talcum powder can reach the ovaries through perineal application (via migration through the female reproductive tract) and through inhalation are unsupported.

In her expert report, Dr. Zelikoff opines that talcum powder can reach the ovaries by means of migration through the female reproductive tract after perineal application as well as through inhalation. Neither opinion is scientifically sound.

Dr. Zelikoff's reproductive tract migration theory would require talc to migrate upwards through the uterus, cervix and fallopian tubes. If this were the case, we would expect to observe an increase in cancers in these respective areas. None, however, has been reported. This has been discussed in detail in Section 11.5.

Nor does the literature cited by Dr. Zelikoff support her reproductive tract theory. Many of the articles Dr. Zelikoff cites regarding reproductive migration do not involve talc at all, but rather analyze migration of other "inert particles." Absent evidence that those "inert particles" share similar migration characteristics with talc, those articles are irrelevant. At her deposition, Dr. Zelikoff could not cite any studies showing that talc particles migrate in the same manner as the particles analyzed in those studies (Zelikoff Deposition, page 426). Dr. Zelikoff also confirmed that she was not aware of any animal or human studies showing that talc migrates from the perineum to the ovaries (Zelikoff Deposition, pages 339-40).

Dr. Zelikoff's inhalation theory is likewise flawed. Dr. Zelikoff opines that "*there is substantial evidence in the scientific and medical literature that support[s] a conclusion that talc powder particles can reach the ovaries through inhalation*" (Zelikoff Report, page 17). That opinion ignores that there is no evidence of an increase in lung cancer or mesothelioma, as would be expected if talcum powder could reach the ovaries through inhalation. It also ignores the fact that no increase in ovarian cancer was seen in occupational exposures to talcum powder, which would be primarily via inhalation and at much higher levels than perineal exposures (Section 7.2).

At deposition, Dr. Zelikoff shifted her theory, opining that biological plausibility was not dependent on talc reaching the ovaries (Zelikoff Deposition, page 298). Dr. Zelikoff could not identify a single study, however, evidencing that talc inhaled through the lungs would cause inflammation in the ovaries that could lead to

ovarian cancer (Zelikoff Deposition, pages 300-03). In fact, the scientific literature generally defines inflammation as a "*response of living tissue to **local** injury…. That it leads to the **local** accumulation of blood cells and fluid*" (emphasis mine) (Ryan and Majno, 1977). The inflammatory response is directed to heal affected tissue, which involves direct interaction with this area, rather than a generalized response in distant tissues (Coussens and Werb, 2002). Thus, talc inhaled through the lungs would cause inflammation in the lungs, not the ovaries.

12.3 Dr. Zelikoff's opinion that talcum powder products cause an inflammatory tissue reaction is similarly unsupported.

Dr. Zelikoff's opinion that talcum powder products cause an inflammatory tissue reaction that may result in ovarian cancer is flawed in several respects. As an initial matter, Dr. Zelikoff's inflammation theory is undermined by scientific studies that anti-inflammatory drugs do not reduce the risk of ovarian cancer (NCI, 2019) and that a history of pelvic inflammatory disease does not increase the risk of ovarian cancer (Merritt, 2008). Her theory is further undermined by the fact that studies have rejected an association between use of talc-dusted diaphragms and condoms and ovarian cancer, as discussed in Section 7.3 (Huncharek et al., 2007, Penninkilampi and Eslick, 2018).

As the sources upon which Dr. Zelikoff relies in her report state, "*the mechanism by which perineal talc may increase the risk of ovarian cancer is uncertain*" (Zelikoff Deposition, page 400). Dr. Zelikoff conceded that "*there is still room for further study*" when it comes to identifying the mechanism linking talc and ovarian cancer (Zelikoff Deposition, page 401). In other words, any opinion about biological mechanism is speculative.

12.4 Conclusion

For the reasons stated above, Dr. Zelikoff's opinion that it is biologically plausible for talcum powder products to cause ovarian cancer is not scientifically sound.

13.0 Additional Methodological Flaws in the Expert Report of Dr. Carson

Dr. Carson does not have a valid scientific basis to conclude that cosmetic talcum powder is carcinogenic or that any association between talcum powder and ovarian cancer is reflective of causation. First, his opinion that talcum powder is carcinogenic flies in the face of IARC's conclusion to the contrary, erroneously assumes that any carcinogen can cause cancer at any dose, and erroneously assumes that any product that causes one type of cancer can cause other types of cancer. These faulty opinions have been addressed in various sections of this report (Sections 5.0 and 6.0). Second, his application of the Bradford Hill factors is fatally flawed. In particular, the consistency of association and strength of association factors on which he purports to place great weight cut ***against*** finding a causal relationship, as discussed in Section 7.3.4 of this report.

13.1 Dr. Carson erroneously concludes that "[t]alcum powder products sold for personal hygiene use are carcinogenic."

Dr. Carson concludes in his report that talcum powder is a complete carcinogen, meaning it can both initiate and promote the development of cancers in the tissues in which it resides. Dr. Carson refers to the production of chronic inflammation and the presence of asbestos, fibrous talc, carcinogenic metals, and fragrant components as supporting this carcinogenic potency. This is in contrast to the IARC classification regarding the perineal use of talcum powder and ovarian cancer, which concluded that the scientific evidence provided "*limited evidence"* of a relationship between perineal talc use and ovarian cancer (IARC, 2018). At his deposition, Dr. Carson acknowledged that "*IARC rejected classification of talc as . . . carcinogenic*" (Carson Deposition, page 225) and that "*IARC's published position is that evidence of a migration theory of talcum powder migrating [to] the ovaries is weak*" (Carson Deposition, page 206). Nevertheless, he insisted that IARC's classification of talc as 2B "is a carcinogenic classification." This conclusion misunderstands the IARC classification system, under which 2B indicates a substance for which there is "*limited evidence*" of carcinogenicity in humans and "*less than sufficient evidence*" in animals (IARC, 2006). Dr. Carson's contention, raised for the first time at his deposition, that IARC is likely to reconsider its classification of talc is based on sources of information such as "*chatter*" (*e.g.*, Carson Deposition, pages 203-04) or social media postings (*see*, *e.g.*, Carson Deposition, page 278), which is not the type of information on which a toxicologist would reasonably rely.

Dr. Carson also erroneously surmises that because a compound is a known human carcinogen, it is 1) sufficiently carcinogenic at any dose, and 2) capable of causing cancer in any tissue. As discussed earlier in this report (Section 6.0), the dose makes the poison. Dr. Carson, however, advances an unscientific "any exposure" theory (Carson Deposition, page 152 (asbestos), page 171 (heavy metals)). He believes in "*a zero threshold approach until we know … a threshold below which exposure would be safe*" (Carson Deposition, page 312). This may be an appropriate precautionary principle, but it is not a scientific way to determine causation. In any event, as discussed, the concentrations of heavy metals, and other allegedly carcinogenic substances, are insufficient to reach a threshold for any adverse health effect, and furthermore, in the case of heavy metals and fragrant chemicals, none have been implicated or associated with ovarian cancer in the scientific literature. Dr. Carson also acknowledges this, stating that certain substances may be associated with some types of cancers and not others (Carson Deposition, page 179).

Dr. Carson also refers to cohort and case-control studies that have shown "statistically significant" associations between talc and ovarian cancer; however, as explained previously, the majority of these studies do not reach statistical significance (Section 7.3). There is high variability in regards to disease sub-type specificity, and very few of these studies showed any dose-response trend or association.

13.2 Dr. Carson's conclusion that "perineal application of talcum powder causes epithelial ovarian cancer in some users and raises the risk of ovarian cancer in all users" misapplies the Hill Criteria.

Dr. Carson opines that the Bradford Hill Criteria support his conclusion that talcum powder causes epithelial ovarian cancer. A few key points include:

- **Strength** – Dr. Carson says that the "*compelling strength of association*" was one of the most important factors in leading him to deduce a causal relationship (Carson Report, pages 9-10). In fact, however, the association is weak and cuts ***against*** finding a causal relationship, as discussed in Section 7.3.4. Carson mentions the odds ratios and relative risk of the various studies examining talc powder usage and ovarian cancer when establishing the strength of the association; however, he only refers to three cohort studies that did not find a significant relative risk and does not address the weak association that all the studies have shown. Even if the relative risk that he calculated – approximately 1.3 – is correct, it is a weak association, not a strong one, as Dr. Carson himself testified (Carson Deposition, pages 232-33). Dr. Carson argued at his deposition that because ovarian cancer is a common disease, if the 1.3 risk ratio were causal, it would represent a significant number of preventable deaths annually (Carson Deposition, page 233). That has nothing to do with whether an association is sufficiently strong to infer causation and shows that Dr. Carson fundamentally misunderstands the Bradford Hill Criteria.

- **Consistency** – Dr. Carson also placed significant weight on the purported consistency of the association between talcum powder use and ovarian cancer. The results, however, are far from consistent. Dr. Carson does not take into account the studies that did not find a statistically significant association between talcum powder use and ovarian cancer. This is especially troubling because not only have many studies failed to show a statistically significant association, but different types of studies have generally produced different results. As Dr. Carson acknowledged at his deposition, "*In applying the Bradford Hill criteria of consistency, there should be consistency across different types of studies*" (Carson Deposition, page 240). But Dr. Carson also acknowledged at his deposition that cohort studies generally do not show a statistically significant association (Carson Deposition, page 238). The difference between cohort studies and case-control studies is significant because case-control studies, unlike cohort studies, are subject to recall bias. Although Dr. Carson "does not believe" that recall bias explains the difference between cohort and case-control study results, he acknowledges that it is a possibility (Carson Deposition, pages 241-42). Dr. Carson also does not address the variation in the scientific literature regarding sub-types of ovarian cancer. While some studies found an increased risk with serous sub-type, others found an increased risk with endometrioid or mucinous sub-types, while others did not differentiate between various sub-types.

- **Specificity** – Dr. Carson states that the "*stimulation of ovarian cancer is particularly associated with the presence of talc in the ovaries and fallopian tubes*" (Carson Report, page 9). Mere presence of a substance in the body does not mean that the substance is responsible for the adverse health affect.

- **Biological Gradient** – While Dr. Carson states that "some" studies have failed to find evidence of a dose-response relationship, he fails to mention that the majority of the literature has not been able to find a dose-response relationship between talcum powder usage and ovarian cancer, as discussed previously, though he acknowledged as much at his deposition (Carson Deposition, page 223, 254-55).

- **Coherence** – Dr. Carson states that the evidence regarding talcum powder is consistent with what is known about other factors related to ovarian cancer such as early menarche, late menopause, late pregnancy or not having children, hormone replacement therapy, etc. Dr. Carson contends that all of these factors can contribute to an inflammatory environment, as can talcum powder usage. But those risk factors are thought to relate to additional ovulation, not inflammation from an external agent. Moreover, Dr. Carson fails to deal with evidence that undermines his coherence theory. For example, he acknowledges that if he is correct that foreign particles can migrate from outside the body up the reproductive tract and cause inflammation and cancer in the ovaries, tubal ligation should decrease the risk of ovarian cancer in perineal talc users by blocking this pathway (Carson Deposition, page 191). However, a recent meta-analysis synthesized the literature on this topic and found that tubal ligation had no effect on the rate of ovarian cancer in perineal talc users (Terry et al., 2013). Moreover, one recent study suggests that the association between talc use and ovarian cancer is ***higher*** among women who have had a tubal ligation or hysterectomy (Cramer et al., 2016).

13.3 Conclusion

Dr. Carson misapplies the Hill Criteria and fails to apply sound toxicological principles, resulting in a highly flawed and unreliable methodology.

14.0 Additional Methodological Flaws in the Expert Report of Dr. Crowley

Dr. Crowley's opinions are not derived from scientific principles of toxicology. First, his regulatory opinions misstate the relevant regulations and fail to consider the concentration of a fragrance chemical necessary to trigger so-called "regulatory concern." Second, he fails to follow appropriate scientific methodology in determining that fragrant chemicals can contribute to potential carcinogenicity because he fails to consider dose-response relationship and fails to differentiate between ovarian cancer specifically and other forms of cancer.

14.1 Dr. Crowley inaccurately opines that the fragrance-related chemicals used in Johnson's Baby Powder and Shower to Shower were not in compliance with government and industry standards.

Dr. Crowley makes several statements in his expert report regarding regulatory compliance, such as:

> "*Several fragrance chemicals do not have an established governmental or industry standard*" (Crowley Report, page 64)
>
> "*Para-cresol is not permitted in cosmetics according to the Cosmetic Ingredient Review Expert Panel"* (Crowley Report, page 64)
>
> "*Of the 53 fragrance chemicals in the product, 9 fragrance chemicals have an exposure limit"* (Crowley Report, page 60)

Dr. Crowley also provides multiple tables showing exposure limits for a subset of fragrant chemicals and whether they are listed or reviewed in various databases or by various agencies, stressing these as chemicals of "regulatory concern." Dr. Crowley 1) misunderstands the relevant regulations, 2) fails to consider the dose at which these exposure limits or regulations apply compared to the concentration of the fragrant chemicals in the relevant products, and 3) ignores the difference between the evidence needed to set a precautionary standard and the evidence needed to demonstrate causation.

First, Dr. Crowley misunderstands the relevant regulations that he claims restrict several of the fragrance chemicals used in the products at issue. He identified a fragrance chemical as being "*of regulatory concern*" if it fell in one of seven categories: "(1*) not listed in Title 21 of the Code of Federal Regulations[;] (2) not approved for fragrance o[r] flavor use[;] (3) not permitted for cosmetic use[;] (4) requires warnings[;] (5) not permitted for use on the body[;] (6) absence of an IFRA standard[;] (7) absence of a CIR listing, or a CIR listing as unsafe or insufficient data to support safety*" (Crowley Report, page 18). As he acknowledged at his deposition, several of these categories, properly understood, do not indicate any "regulatory concern." For example, fragrance chemicals need not be listed in Title 21, and the absence of a listing only indicates that the FDA "*has not reviewed data*" "*one way or the other*" (Crowley Deposition, page 154). The CIR similarly does not review all fragrance chemicals and, in particular, does not bother to review fragrance chemicals if their safety has already been determined by the Research Institute for Fragrance Materials, such that no inference can be drawn from the mere lack of CIR review. Perhaps most strikingly, the absence of an International Fragrance Association (IFRA) standard ***supports*** a finding of safety, for such standards are only issued when "the safety assessment ***does not*** support current use" (emphasis mine) (Crowley Deposition, pages 159-160).

Dr. Crowley's report also misapprehends the regulations around certain specific fragrances. For example, he contends that Myroxylon Pereirae oil, commonly known as balsam peru oil, is prohibited by both the IFRA and the European Union. As he acknowledged at his deposition, however, he had confused balsam Peru oil with balsam peru crude (Crowley Deposition, page 164-68). Balsam peru ***oil*** is not prohibited by IFRA, and is "Generally recognized As Safe (GRAS)" by the FDA (FDA, 2018c). In a similar vein, Dr. Crowley claims that ethenylbenzene, commonly known as styrene, is no longer approved by the FDA for use as a flavor or fragrance. However, ethenylbenzene is still approved for use as a fragrance and was only delisted as a flavoring because it is no longer used (FDA, 2018b).

Second, Dr. Crowley's regulatory opinions fail to acknowledge that many of the restrictions that he cites apply only to use of a fragrance chemical above a given concentration. For example, IFRA has set exposure limits for many chemicals, but concluded that those chemicals can safely be used in lower concentrations. Similarly, the FDA allows the use of some chemicals that are classified as GRAS, and allows others as residues up to a designated concentration as a result of production and/or packaging (FDA, 2018a). Because Dr. Crowley failed to determine the concentration or amount of any fragrant chemical in the talcum powder products at issue, even though the Johnson & Johnson defendants produced the information necessary to do so, he did not and could not determine whether the amount exceeded any regulatory threshold.

Finally, even if Dr. Crowley could show that the amount of certain fragrant chemicals in the talcum powder products at issue exceeded a regulatory threshold, his regulatory opinions would still not be the type of evidence that toxicologists consider when determining causation. As discussed at greater length above, because regulations are generally set out of an abundance of caution and in order to preserve a margin of safety, they are not helpful in evaluating a potential causal relationship.

14.2 Dr. Crowley does not follow accepted scientific principles to reach his opinion that fragrant chemicals contribute to potential carcinogenicity.

Dr. Crowley's conclusion that fragrant chemicals potentially contribute to the purported carcinogenicity of talcum powder ignores causation criteria, the dose-response principle and the low concentration of fragrances in the products at issue and ignores the fact that even if certain chemicals may be associated with some other cancers, none of them is associated with ovarian cancer specifically.

First, Dr. Crowley ignores dose-response and the concentration of products in his assessment. As stated earlier in this report, "*All substances are poisons; there is none which is not a poison. The right dose differentiates a poison from a remedy*" (Klaassen, 2013). Thus, to ignore the role of dose, exposure, and endpoint is to ignore the central tenets to understanding how substances become poisons. As such, the classification of a chemical as carcinogenic, irritating, corrosive, or otherwise harmful is based upon, among other things, dose. Dr. Crowley acknowledged as much at his deposition (Crowley Deposition, page 129).

Dose and concentration play vital roles in the classification of mixtures and compounds as irritants, sensitizers or potential carcinogens, as previously discussed. The fact that a chemical has a critical effect or an exposure limit does not mean that 1) any exposure is harmful, and 2) all exposures can contribute to an adverse health effect. As previously discussed, the publicly available GHS has guidelines regarding the classification of compounds and mixtures as irritants based upon the dose of exposure and the endpoint; without that information, we cannot deduce whether these fragrant chemicals are present at high enough concentrations to cause irritation. In his deposition, Dr. Crowley erroneously refers to GHS hazard codes as "classifications" (Crowley Deposition, page 303-04). Dr. Crowley then stated that he did not review the GHS or underlying science, but merely reviewed material safety data sheets (MSDSs), company websites, and website such as Google Scholar for his assessment (Crowley Deposition, pages 303-05). As previously mentioned, the GHS is freely available on the OSHA Hazard Communication Standard website. These hazard statements quoted by Dr. Crowley are based upon a metric assessing scientific research and concentrations of compounds within a mixture, as previously described. In other words, a known irritant must be present in a mixture above a certain percentage (0.1%) in order for the mixture to be classified as an irritant and warrant the aforementioned hazard statements. As shown in Appendix D, none of the fragrant components of the talcum powder products at issue would meet these criteria.

Nevertheless, Dr. Crowley makes no reference to the importance of dose when assessing the fragrant chemicals in the products at issue. For example, in Table 7 of Dr. Crowley's report (beginning on page 22), he identifies whether the fragrant chemical is listed in the RTECS Database and notes some of the toxicity

concerns, many of which have a dose associated with them. Dr. Crowley does not provide the dose information for all chemicals, but the ones that do show the dose associated with the toxicity concern are not put into the context of the concentration of the material in the products at issue.[5] Dr. Crowley repeats this process in his assessment of whether the fragrant chemicals have been listed as irritants, skin irritants, and eye irritants in Table 8 of his report (Crowley Report, page 28), and in his assessment of sensitization (Crowley Report, Table 9, page 33), allergens and dermatitis (Crowley Report, Table 10, page 35), critical effects (Crowley Report, Table 11, page 37), and throughout his report.

For comparison, a summary table comparing the concentration of the fragrant chemicals as provided in the proprietary information from the Johnson & Johnson defendants is compared with the appropriate exposure limits and guidelines that he also discusses in Appendix D (Proprietary-Attorney eyes only). When compared to concentration and dose information as shown in Appendix D, none of the fragrant chemicals is present at a concentration where the normal use of the products at issue would result in an adverse health effect.

Dr. Crowley merely states whether the compound has been listed as an irritant, not the dose or concentration at which the irritation occurs. In fact, Dr. Crowley testified at his deposition that he did not even know, much less consider, the amount of any fragrant chemical contained in the talc powders at issue, or the amount to which a woman who used the talc powders at issue would be exposed (Crowley Deposition, pages 107, 201, 302). Without this information, Dr. Crowley can make no deductions as to whether a fragrant chemical or other compound present in Johnson's Baby Powder or Shower to Shower would be in compliance with governmental standards, or whether they would present a hazard to the consumer, as he admits (Crowley Deposition, page 106).

Because Dr. Crowley did not consider the concentration of fragrant chemicals in the talcum powder products at issue, he did not consider the amount to which any user of the powders would be exposed. As such, he has performed no dose response, exposure assessment, or risk assessment to support his opinions that the fragrant chemicals contribute to the inflammatory properties, toxicity and potential carcinogenicity of talcum powder.

Although Dr. Crowley generally acknowledged the principle that the dose makes the poison at his deposition, he also contended, for the first time, that for certain genotoxic chemicals, "[*o]ne molecule is enough to cause an increased risk*" (Crowley Deposition, page 125). This is contrary to the scientific consensus. As previously discussed in Section 6.3, genotoxicity, as with any toxic endpoint, is frequently dose-associated. Indeed, while a genotoxin may cause DNA damage at low levels, or assume a linear dose-response curve, this is based upon the mechanism of action, target cells, genes affected, etc.

[5] At Dr. Crowley's deposition, he contended repeatedly that he failed to calculate concentration or exposure data because he had not been provided with sufficient information from defendants. However, as mentioned above, it is my understanding that plaintiffs' counsel were provided with three proprietary documents, including Exhibit 1: Johnson's Baby Powder Fragrance Ingredients, Exhibit 2: Shower to Shower Fragrance Ingredients, and Exhibit 3: Changes to Johnson's Baby Powder Fragrance Ingredients, along with two Formula Declaration Reports (JNJTALC000891091 and JNJTALC000149667). From these same documents, I was able to calculate the concentrations of fragrant chemicals set forth in Appendix D.

14.3 Dr. Crowley fails to consider the relationship between fragrant chemicals and ovarian cancer specifically.

As stated earlier, none of the fragrant chemicals reported to be present in the products at issue has been associated with ovarian cancer. It is fundamental that a substance may be associated with certain forms of cancer and not with other forms of cancer, as Dr. Crowley agrees (Crowley Deposition, page 212). Dr. Crowley further agreed that it is possible for a substance to cause or contribute to the development of cancer in an animal, but not in humans (Crowley Deposition, pages 212-13). While Dr. Crowley refers in his report and deposition to some studies showing a relationship between high doses of certain fragrant chemicals and some forms of ovarian cell toxicity, primarily in Chinese hamster ovary cell models, none of them shows an increase in rates of ***ovarian*** cancer (Crowley Deposition, pages 114-15, 217-18). In addition, these studies are not *in situ,* or animal studies, but rather cell-line, *in vitro*, studies where a concentration of the fragrant chemical was directly added to the media covering the cells in a petri dish. Dr. Crowley himself later conceded that he had not found a publication that linked the fragrance chemicals in Johnson's Baby Powder and Shower to Shower to human ovarian cancer (Crowley Deposition, pages 114-15). These cell-line studies remove the natural processes in the body, including inflammation and repair mechanisms, and reduce the experiment to one cell type, whereas many different types of cells are involved in the female reproductive and immune systems. As previously discussed in Section 6.1, *in vitro* studies are useful to understand potential mechanisms of toxicity; however, they cannot be used to determine dose-response, or indeed to indicate whether an actual response in living tissue will occur, due to the extremely artificial nature of these types of experiments.

14.4 Conclusion

In summary, Dr. Crowley has no established methodology for his conclusions, as he stated that he performed no dose-response assessment, no exposure assessment, and no risk assessment. Dr. Crowley's lack of methodology contravenes the scientific principles of toxicology, because it ignores the importance of dose and the possibility that a substance might be associated with certain cancers, but not others. It is not a valid methodology for assessing risk; is not backed by regulatory bodies; nor is it based in sound science. As such, Dr. Crowley's conclusions and opinions are not scientifically sound.

15.0 Lack of Attribution and Reliance on Non-Scientific Sources

Plaintiffs' experts' reports are also methodologically flawed because they did not identify sources for many of their statements and relied on non-scientific sources. For example, page 20 of Dr. Zelikoff's report contains the following passage:

> *"Both inherited and acquired gene mutations work together to cause cancer. Even if one has inherited a genetic mutation that predisposes one to cancer, that doesn't mean he or she is certain to get cancer. Rather, one or more additional gene mutations may be needed to cause cancer. The inherited gene mutation could instead make one more likely to develop cancer when exposed to certain cancer-causing substances"* (Zelikoff Report, page 20).

Dr. Shawn Levy's report contains the following paragraph on page 5:

> *"**Both inherited and acquired gene mutations work together to cause cancer.** While genetic testing has become commonplace for both assessing risk for cancer as well as directing treatment, the catalog of oncogenes, tumor suppressor genes, and DNA repair genes make genetic testing valuable and impactful for informing patients of their genetic risk for cancer. Genetic testing generally detects inherited mutations. Currently, genetic screening does not detect acquired gene mutations because they occur only in certain cells. **Even if one has inherited a genetic mutation that predisposes one to cancer, that doesn't mean he or she is certain to get cancer. Rather, one or more additional gene mutations may be needed to cause cancer. The inherited gene mutation could instead make one more likely to develop cancer when exposed to a certain cancer-causing substance**"* **(emphasis mine)** (Levy Report, page 5).

When asked to explain how the exact same words appeared in her report and the report of another expert ***that she claims to not have previously seen***, Dr. Zelikoff stated that it was her practice to "*use[] sentences from . . . other people's papers because they were common knowledge and contributed by multiple authors*" without citation (Zelikoff Deposition, page 83).

Similarly, Dr. Crowley's report contains the following paragraph on page 18:

> *"Regulation of consumer products largely falls under the Consumer Product Safety Act ("CPSA"). The CPSA does not require disclosure of all ingredients in products. Instead of listing ingredients, a manufacturer can provide other information on a product, such as a warning label. Similarly, the Federal Hazardous Substances Act (FHSA) requires warning labels for hazardous substances, but does not require that all ingredients be disclosed on the product's label. Ingredients can also be exempt from disclosure through "trade secrets" protection. Under the FFDCA, fragrance ingredients that qualify as trade secrets may be listed as "and other ingredients" without disclosing the ingredients*" (Crowley Report, page 18).

Dr. Anne Steinemann, in an article titled "*Fragranced consumer products and undisclosed ingredients"* that was published in the Environmental Impact Assessment Review, wrote the following:

> ***"Regulation of consumer products** (other than food, drugs, cosmetics, tobacco, and pesticides) **largely falls under the Consumer Product Safety Act (CPSA)**. . . . Notably, **the CPSA does not require disclosure of all ingredients in products. Instead of listing ingredients, a manufacturer can provide other information on a product, such as a warning label. Similarly, the Federal Hazardous Substances Act (FHSA) requires warning labels for hazardous substances, but does not require that all ingredients be disclosed on the product's label.***
>
> ***Ingredients can also be exempt from disclosure through "trade secrets" protection. . . Under the FFDCA, fragrance ingredients that qualify as trade secrets may be listed as***

"and other ingredients," without disclosing the ingredients" (emphasis mine) (Steinemann, 2009)**.**

On page 32 of his report, Dr. Crowley appears to have copied verbatim a paragraph about sensitization from a website called Interactive Learning Paradigms, Incorporated (Crowley Report, page 32; Crowley Deposition, pages 102-104).

Perhaps the most troubling example is on page 27 of Dr. Crowley's report, which states as follows:

> *"A mucous membrane or mucosa is a membrane that lines various cavities in the body and covers the surface of internal organs. It consists of one or more layers of epithelial cells overlying a layer of loose connective tissue. It is mostly of endodermal origin and is continuous with the skin at various body openings such as the eyes, ears, inside the nose, inside the mouth, lip, vagina, the urethral opening, and the anus. Some mucous membranes secrete mucus, a thick protective fluid. The function of the membrane is to stop pathogens and dirt from entering the body and to prevent bodily tissues from becoming dehydrated"* (Crowley Report, page 27).

The first paragraph of the Wikipedia entry for "mucous membrane" reads as follows:

> ***"A mucous membrane or mucosa is a membrane that lines various cavities in the body and covers the surface of internal organs. It consists of one or more layers of epithelial cells overlying a layer of loose connective tissue. It is mostly of endodermal origin and is continuous with the skin at various body openings such as the eyes, ears, inside the nose, inside the mouth, lip, vagina, the urethral opening and the anus. Some mucous membranes secrete mucus, a thick protective fluid. The function of the membrane is to stop pathogens and dirt from entering the body and to prevent bodily tissues from becoming dehydrated"*** *(emphasis mine)***.**

Of course, Wikipedia is not a reliable scientific source – it can be edited by anyone and, without further examination of any sources cited in the Wikipedia article itself, it is impossible to know whether the information there is accurate.

These sorts of practices are troubling and call into question the reliability of the reports as a whole. Although scientists naturally build upon one another's work, direct copying of another scientist's words or ideas without proper citation is intellectually dishonest (Masic, 2012, Kumar et al., 2014). In a world with increasingly easy digital access to the works of others, we must be even more vigilant to properly cite and quote the sources we use. After all, as Masic put it, "*[t]ruth and trustworthy results are 'flesh and bones' of scientific research… The worst forms of scientific misconduct and intellectual dishonesty are… [p]lagiarism of ideas and words (stealing others' ideas, data, texts)*" (Masic, 2012).

16.0 Conclusions

In conclusion, the scientific evidence put forth by plaintiffs' experts in an attempt to prove a causal relationship between the exposure to the talcum powder products at issue and ovarian cancer does not meet the methodological standards and criteria for causality. The evidence regarding causation, the presence of heavy metals, asbestos, and fragrant chemicals, and toxicity information do not support that the perineal or inhalation exposure to talcum powder significantly increases an individual's risk of ovarian cancer.

All of my opinions as stated above are to a reasonable degree of toxicological and scientific certainty. I reserve the right to amend this report if new information becomes available.

Respectfully,

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17.0 References

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APPENDIX A

Documents Received and/or Reviewed by Dr. Scribner Tuttle

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Documents Received and/or Reviewed by Dr. Scribner Tuttle

- Expert Report and Deposition of Dr. Crowley
- Expert Report and Deposition of Dr. Longo and Dr. Rigler
- Expert Report of Dr. Kane
- Expert Report of Dr. Alan Campion
- Expert Report of Dr. McTiernan
- Expert Report of Dr. Zambelli-Weiner
- Expert Report and Deposition of Dr. Carson
- Expert Report of Dr. Clarke-Pearson
- Expert Report of Dr. Kessler
- Expert Report of Dr. Smith
- Expert Report of Dr. Siemiatycki
- Expert Report of Dr. Wolf
- Expert Report and Deposition of Dr. Zelikoff
- Expert Report and Deposition of Dr. Plunkett
- Expert Report of Dr. Krekeler
- Expert Report of Dr. Moorman
- Expert Report of Dr. Smith-Bindman
- Expert Report of Dr. Cook
- Expert Report of Dr. Levy
- Expert Report of Dr. Singh
- Expert Report of Dr. Saed
- IMERYS and Johnson & Johnson Defendants' Documents cited in Dr. Cook and Dr. Krekeler Reports
- Exhibit 1 – Attorneys' Eyes Only – Johnson's Baby Powder Fragrance Ingredients
- Exhibit 2 – Attorneys' Eyes Only – Shower to Shower Fragrance Ingredients
- Exhibit 3 – Attorneys' Eyes Only – Changes to Johnson's Baby Powder Fragrance Ingredients
- JNJTALC000891091 – Formula Declaration Report
- JNJTALC000149667 – Formula Declaration Report
- Scientific Literature (see references)

APPENDIX B

Curriculum Vitae of

Kelly Scribner Tuttle, Ph.D., CIH

CTEH®

THE SCIENCE OF READY℠

KELLY SCRIBNER TUTTLE, Ph.D., CIH

Senior Toxicologist

ktuttle@cteh.com

INTRODUCTION

Dr. Kelly Scribner Tuttle is a Senior Toxicologist at the Center for Toxicology and Environmental Health, L.L.C. (CTEH®). Dr. Scribner Tuttle has experience in the fields of chemical emergency response, human and environmental toxicology, cell biology, crisis communication, human health and ecological risk assessment, vapor instrusion, systems biology, physiology, and cancer research. She received her Ph.D. in the Philosophy in Toxicology from the Texas A&M University. Upon completion of her fellowship in 2013, Dr. Scribner Tuttle accepted a position as a Toxicologist at CTEH® in Little Rock, AR. Dr. Scribner Tuttle is a board Certified Industrial Hygienist (CIH) through the American Board of Industrial Hygiene and a member of the American Industrial Hygiene Association, the American Conference of Governmental Industrial Hygienists, and the Society of Toxicology.

Dr. Scribner Tuttle participates in a variety of projects with scopes ranging from chemical product evaluation and dose reconstruction to environmental contamination, vapor intrusion, and human health and ecological risk assessment. Dr. Scribner Tuttle also participates in the Toxicology Emergency Response Program, leading air monitoring and environmental sampling teams to address worker and public safety after hazmat incidents across the country. She is consulted by clients for her expertise in worker chemical exposure incidents and is asked to convey toxicological information to workers, supervisors, and health care providers alike to improve the communication of health risks to workers and employers and the quality of toxicological information used by treating physicians. She is also called by government agencies as well as hazardous materials shipping, handling, and manufacturing and petroleum industry clients to provide expert toxicological and human health risk support in emergency situations where releases of hazard materials pose a threat to workers, residents, and the environment, as well as in industrial and operational settings. She also has expertise in emergency preparedness and planning and is a trusted partner of industry and governments alike in emergency response management and safety.

EDUCATION

Ph.D., Toxicology
Texas A&M University
College Station, TX

B.S., Veterinary Science
University of Nebraska
Lincoln, NE

PROFESSIONAL AFFILIATIONS

Society of Toxicology

- Society of Toxicology - Occupational and Public Health Specialty Section
- Society of Toxicology - Regulatory and Safety Evaluation Specialty Section
- Society of Toxicology - Carcinogenesis Specialty Section
- Society of Toxicology - Lone Star Regional Chapter

American Industrial Hygiene Association

- American Industrial Hygiene Association - North Texas Chapter

American Conference of Governmental Industrial Hygienists

REGISTRATIONS AND CERTIFICATIONS

- Certified Industrial Hygienist #CP 11510
- BOSIET
- 40-Hour HAZWOPER Training
- TWIC Card

AWARDS & HONORS

1. TAMU Auxiliary Graduate Student Award, Texas A&M University, College Station TX, April, 2013.

2. High Impact Research Achievement Award, First Author Publication, Texas A&M University, College Station TX, April, 2013.

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3. Ethel Ashworth-Tsutsui Memorial Award for Research – Women in Science and Engineering, Texas A&M University, College Station TX, 2012.

4. High impact Research Achievement Award, Extramural Funding, Texas A&M University, College Station TX, April, 2012.

5. TAMU Academic Excellence Award - Texas A&M University, College Station TX, April, 2012.

6. George T. Edd's Outstanding Toxicology Student Award – TAMU Interdisciplinary Faculty of Toxicology, Texas A&M University, College Station TX, April, 2012.

7. CVM Graduate Student Research Trainee Grant - College of Veterinary Medicine and Biosciences, Texas A&M University, College Station TX, 2011.

8. CVM Award for Exemplary Work during Graduate Career - College of Veterinary Medicine and Biosciences, Texas A&M University, College Station TX, 2011.

9. DOD BCRP Pre-Doctoral Traineeship Award – Department of Defense Congressionally Directed Medical Research Program, January, 2011.

10. San Antonio Breast Cancer Symposium Basic Science Scholars-In-Training Award – American Association for Cancer Research, San Antonio TX, December, 2010.
11. TAMU Toxicology Regents Fellowship - TAMU Interdisciplinary Faculty of Toxicology, Texas A&M University, College Station TX, August, 2008.

EMERGENCY MANAGEMENT AND CRISIS PREPAREDNESS EXPERIENCE

Invited Participant - IMT WCD Exercise - Bellingham, WA, 2018

Invited Participant - IMT Table Top Exercise - Nikiski, AK, 2017

Invited Participant - Long Beach WCD Exercise - Long Beach, CA, 2016

Invited Participant - Merritt/Kamploops Full Scale Exercise - Kamloops, BC, CA, 2016

Invited Participant - San Francisco Bay WCD Exercise - Burlingame, CA, 2016

Invited Participant - USCG Sector Delware Bay Ecological Risk Assessment - Sharon Hill, PA, 2015

Invited Participant - Westridge Terminal Exercise - Coquitlam, BC, 2015

Invited Participant - Pipeline Release Table Top Exercise - Grant County, AR, 2015

Invited Participant - Pipeline Release Table Tope Exercise - Poplar Bluff, MO, 2014

PUBLICATIONS

Refereed Publications:

1. Berg, M., Scribner, K., Harrill, J., Goad, P., Still, K., Hesterberg, T., 2018. "Toxicology of Diesel Particulate Matter." In: Luttrell, B., Still, K., Church, J. (Ed.), Toxicology Principles for Industrial Hygienists, 2nd edition. American Industrial Hygiene Association (AIHA). (In Press).

2. Scribner, K., Kind, J., Nony, P., Still, K., Hesterberg, T., 2018. "Toxicology of Asbestos." In: Luttrell, B., Still, K., Church, J. (Ed.), Toxicology Principles for Industrial Hygienists, 2nd edition. American Industrial Hygiene Association (AIHA). (In Press).

3. Pearson, S.J., Tapasree, R.S., McQueen, C.M., Elswood, J., Schmitt, E.E., Wall, S.W., Scribner, K.C., Wyatt, G., Barhoumi, R., Behbod, F., Rijnkels, M., Porter, W.W. (2018) "ATM-dependent activation of SIM2s regulates homologous recombination and epithelial-mesenchymal transition." Oncogene, Published online 10 December, 2018.

4. Nony, P., Scribner, K., Hesterberg, T. (2014) "Synthetic Vitreous Fibers." In: Wexler, P. (Ed.), Encyclopedia of Toxicology, 3rd Editions Vol. 4. Elsevier Inc., Academic Press, pp. 448-453.

5. Scribner K.C., Behbod F., and Porter W.W. (2013) "Regulation of DCIS to invasive breast cancer progression by Singleminded- 2s (SIM2s)." Oncogene, May;32(21):2631-9.

6. Romoser A.A., Figueroa D.E., Sooresh A, Scribner K.C., Chen P.L., Porter W.W, Criscitiello M.F., and Sayes C.M.(2012) "Differential NF-kB competency mediates nanoparticle toxicity in normal human dermal cells." Toxicology Letters, May 5;210(3):293-301.

7. Scribner K.C., Wellberg E.A., Metz R.P., Porter W.W. (2011) "Singleminded-2s (Sim2s) promotes delayed involution of the mouse mammary gland through inhibition of Stat3 and NFkB." Molecular Endocrinology, Apr;25(4):635-44.

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PRESENTATIONS

1. Toxicology of Silica, 2018 AIHce Conference - Professional Development Course: Toxicology of Particulate Matter, May 2018, Philadelphia, PA.

2. Toxicology of Asbestos, 2018 AIHce Conference - Professional Development Course: Toxicology of Particulate Matter, May 2018, Philadelphia, PA.

3. Public Health Response to Oil Spills, 2018 Stakeholder Meeting: Prioritizing Public Health Risks from Oil Spills, April 2018, College Station, TX.

4. In-Situ Chemical Oxidation of Ethanol in Groundwater, 2017 AHMP National Conference, August 2017, Fort Worth, TX.

5. Vapor Intrusion and Chlorinated Solvents in Commercial and Industrial Settings, 2017 AHMP National Conference, August 2017, Fort Worth, TX.

6. HazMat Toxicology and Air Monitoring for First Responders, 2017 BRAMAS Haz Mat School, March 2017, Baton Rouge, LA.

7. Applied Toxicology and Risk Assessment in Emergency Response, Texas A&M University Toxicology Lecture Series, February 2017, College Station, TX.

8. The Global Harmonization Standard and the Maritime Industry, New Orleans U.S. Coast Guard Training, January 2017, New Orleans, LA.

9. The Global Harmonization Standard and the Maritime Industry/ Potential Environmental Impacts of the Maritime Industry not related to spills, New Orleans WISTA Monthly Meeting, OCtober 2016, New Orleans, LA.

10. Toxicology of Vapor Intrusion, Mississippi Department of Environmental Quality Introduction to Vapor Intrusion, May 2016, Jackson, MS.

11. Toxicology in Railroads and Emergency Response, BNSF-UT Southwestern Toxicology Residents Meeting, April 2016, Fort Worth, TX.

12. The Use of an Activity Based Study (ABS) as a Novel Method for Tracing Mercury Contamination Platform, American Industrial Hygiene Conference and Exposition, American Industrial Hygiene Association, June 2015, Salt Lake City, UT.

13. Health and Safety Concerns Associated with Response to Crude Oil Releases, USCG Sector Delaware Bay Ecological Risk Assessment Workshop for Bakken and Dilbit Crude Oils, June 2015, Sharon Hill, PA.

14. Risk Assessment: Untangling the Web Platform, American Occupational Health Conference, American College of Occupational and Environmental Medicine, April 2014, San Antonio, TX.

15. Potential Exposure Threshold of Chrysotile Asbestos Poster, Society of Toxicology Annual Meeting, March 2014, Phoenix, AR.

16. Singleminded-2s inhibits DCIS progression by regulating senescence-dependent metabolic equilibrium Poster, Society of Toxicology Annual Meeting, March 2013, San Antonio, TX.

17. Mediation of a Metabolic "Switch" from DCIS to IBC Platform, 8th Annual Breast Cancer Research and Education Program, September 2012, Montgomery, TX (3rd Place).

18. Singleminded-2s (Sim2s) plays a unique role in mammary gland and breast cancer autophagy and energy homeostasis Poster, American Association for Cancer Research Special Conference on Metabolism in Cancer, October 2011, Baltimore, MD.

19. Singleminded-2s (Sim2s) plays a unique role in mammary gland and breast cancer autophagy and metabolism homeostasis Poster, 7th Annual Breast Cancer Research and Education Program, September 2011, Montgomery, TX.

20. Singleminded-2s (Sim2s) induces metabolic and autophagic changes in the functioning mammary gland and breast cancer Poster, Mammary Gland Biology Gordon Research Conference, Salve Regina University, June 2011, Newport, RI.

21. Singleminded-2s (Sim2s) in Breast Cancer Seminar, Interdisciplinary Faculty of Toxicology, Texas A&M University, February 2011, College Station, TX.

22. Inhibition of DCIS progression through promotion of differentiation Poster, CTRC-AACR Breast Cancer Research Symposium, December 2010, San Antonio, TX.

23. Singleminded-2s (Sim2s) inhibits MCF10DCIS.COM progression in vivo by promoting differentiation Platform, Gulf Coast Society of Toxicology Annual Meeting, October 2010, Houston, TX (2nd Place).

24. Singleminded-2s (Sim2s) inhibits MCF10DCIS.COM progression and metastasis in vivo by promoting differentiation Platform, 6th Annual Breast Cancer Research and Education Program, September 2010, Montgomery, TX.

25. Singleminded-2s (Sim2s) inhibits MCF10DCIS.COM progression and metastasis in vivo by promoting differentiation Poster, MRS-AACR Metastasis and the Tumor Microenvironment Conference, September 2010, Philadelphia, PA.

26. Singleminded-2s (Sim2s) delays apoptosis and involution of the mammary gland through inhibition of phospho-Stat3 Poster, Texas Forum on Reproductive Sciences, April 2010, Houston, TX (3rd Place).

27. MDF10DCIS.COM Breast Cancer Cell Lines are Attenuated by Expression of Singleminded- 2s(Sim2s) Platform, Gulf Coast Society of Toxicology Annual Meeting, October 2009, Austin, TX.

28. Aberrant Involuting Pathways in MMTV-Singleminded-2s (Sim2s) Mice Poster, 5th Annual Breast Cancer Research and Education Program, September 2009, Houston, TX.

29. Differential Induction of Involution in MMTV Singleminded-2s (Sim2s) Mice Poster, Mammary Gland Biology Gordon Research Conference, June 2009, Newport, RI.

APPENDIX C

IMERYS AND JOHNSON & JOHNSON Documents cited by Dr. Cook and Dr. Krekeler

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051370	V-97	X-Ray Diffraction	Amphibole Chrysotile	NQ* ND*	ND: Not detected NQ: The concentration of mineral is less than 0.1% and was not quantified *: XRD results were corroborated by polarized light microscopy The amphibole found in V-97 was determined by PLM to be actinolite in the form of cleavage fragments Monthly Composite: May 1992
IMERYS 051371 IMERYS 051431	Float Feed	X-Ray Diffraction	Amphibole Chrysotile	NQ* ND	ND: Not detected NQ: The concentration of mineral is less than 0.1% and was not quantified *: XRD results were corroborated by polarized light microscopy The amphibole found in Float Feed (92-12) was determined by PLM to be actinolite in the form of cleavage fragments Monthly Composite: CWM 92-12; April 1992
IMERYS 051371 IMERYS 051431	Grade 36	X-Ray Diffraction	Amphibole Chrysotile	ND* ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy Monthly Composite: CWM 92-13; April 1992
IMERYS 051371 IMERYS 051431	Beta & Gamma	X-Ray Diffraction	Amphibole Chrysotile	ND* ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy Monthly Composite: CWM 92-14; April 1992
IMERYS 051371 IMERYS 051431	66/96 Product	X-Ray Diffraction	Amphibole Chrysotile	ND* ND	ND: Not detected *: XRD results were corroborated by polarized light microscopy Monthly Composite: CWM 92-15; April 1992

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051371 IMERYS 051431	Float Feed	X-Ray Diffraction	Amphibole Chrysotile	NQ* ND	ND: Not detected NQ: The concentration of mineral is less than 0.1% and was not quantified *: XRD results were corroborated by polarized light microscopy The amphibole found in Float Feed (92-16) was determined by PLM to be actinolite in the form of cleavage fragments Monthly Composite: CWM 92-16; May 1992
IMERYS 051371 IMERYS 051431	Grade 36	X-Ray Diffraction	Amphibole Chrysotile	NQ* ND*	ND: Not detected NQ: The concentration of mineral is less than 0.1% and was not quantified *: XRD results were corroborated by polarized light microscopy The amphibole found in Grade 36 (92-17) was determined by PLM to be actinolite in the form of cleavage fragments Monthly Composite: CWM 92-17; May 1992
IMERYS 051371 IMERYS 051431	Beta & Gamma	X-Ray Diffraction	Amphibole Chrysotile	NQ* ND*	ND: Not detected NQ: The concentration of mineral is less than 0.1% and was not quantified *: XRD results were corroborated by polarized light microscopy The amphibole found in Beta & Gamma (92-18) was determined by PLM to be actinolite in the form of cleavage fragments Monthly Composite: CWM 92-18; May 1992
IMERYS 051371 IMERYS 051431	66/96 Product	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Monthly Composite: CWM 92-19; May 1992

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051372 IMERYS 051432	C-1	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy Daily Composite: 10/24/90
IMERYS 051372 IMERYS 051432	C-2	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy Daily Composite: 10/24/90
IMERYS 051372 IMERYS 051432	C-1	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy Daily Composite: 10/25/90
IMERYS 051372 IMERYS 051432	C-1	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy Daily Composite: 10/26/90
IMERYS 051372 IMERYS 051432	C-1	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy Daily Composite: 10/27/90
IMERYS 051372 IMERYS 051432	C-1	X-Ray Diffraction	Amphibole Chrysotile	ND* ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy Daily Composite: 10/29/90
IMERYS 051372 IMERYS 051432	# 3 Float Feed	X-Ray Diffraction	Amphibole	ND	ND: Not detected Weekly Composite: Week of 10/23/90
IMERYS 051372 IMERYS 051432	# 2 Float Feed	X-Ray Diffraction	Amphibole	ND	ND: Not detected Weekly Composite: Week of 10/23/90
IMERYS 051372 IMERYS 051432	C-0	X-Ray Diffraction	Amphibole	ND	ND: Not detected Weekly Composite: Week of 10/23/90
IMERYS 051372 IMERYS 051432	V-300	X-Ray Diffraction	Amphibole	ND	ND: Not detected Weekly Composite: Week of 10/23/90

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051372 IMERYS 051432	V-6	X-Ray Diffraction	Amphibole	ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy Weekly Composite: Week of 10/23/90
IMERYS 051372 IMERYS 051432	V-700	X-Ray Diffraction	Amphibole	ND	ND: Not detected Weekly Composite: Week of 10/23/90
IMERYS 051372 IMERYS 051432	C-2	X-Ray Diffraction	Amphibole	ND	ND: Not detected Weekly Composite: Week of 10/23/90
IMERYS 051373 IMERYS 051433	Float Feed	X-Ray Diffraction	Amphibole Chrysotile	ND* ND	ND: Not detected *: XRD results were corroborated by polarized light microscopy No asbestiform amphibole minerals were detected Monthly Composite: LAI 92-36; September 1992
IMERYS 051373 IMERYS 051433	Grade 36	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy No asbestiform amphibole minerals were detected Monthly Composite: LAI 92-37; September 1992
IMERYS 051373 IMERYS 051433	Beta & Gamma	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy No asbestiform amphibole minerals were detected Monthly Composite: LAI 92-38; September 1992
IMERYS 051373 IMERYS 051433	W. W. Product	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected *: XRD results were corroborated by polarized light microscopy No asbestiform amphibole minerals were detected

Bates #	Product Tested	Method	Analytes	Results	Comments
					Monthly Composite: LAI 92-39; September 1992
IMERYS 051374 IMERYS 051434	W. W. Product	X-Ray Diffraction	Amphibole Serpentine	ND ND	ND: Not detected XRD results were verified by polarized light microscopy (dispersion staining method) No asbestiform material was detected in either sample, nor was any form of amphibole or serpentine present Grade 66 Silo 1: 10/5/94 - 10/10/94 Grade CO+ (High Bulk) Silo 7: 9/29/94 - 10/5/94
IMERYS 051413	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Grade 66 Silo 3: March 2 - 5, 1998 Grade 66 Silo 2: March 25 - 29, 1998 Reference: A98127
IMERYS 051413	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Grade 66 Silo 4: March 29 - April 2, 1998 Grade 66 Silo 1: April 2 - 8, 1998 Reference: A98127

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051414	Grade 66	Polarized Light Microscopy	Chrysotile	ND	ND: Not detected No asbestiform minerals were detected Grade 66 Silo 1 Composite at West Windsor Reference: A98071
IMERYS 051415	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Grade 66 Silo 4: February 15 - 19, 1998 Grade 66 Silo 1: February 25 - March 2, 1998 Reference: A98072
IMERYS 051416	Grade 66	Johnson & Johnson Test No. TM7024 ("Analysis of Powdered Talc for Asbestiform Minerals by Transmission Electron Microscopy," REV: 08/21/95)	Total Asbestos	N/D	N/D: None detected Grade 66 Composite: 4th Quarter 1997 Reference: 97-448
IMERYS 051417	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Grade 66 Silo 1: January 22 - 26, 1998 Grade 66 Silo 2: February 11 - 15, 1998 Reference: A98047

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051418	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Grade 66 Silo 3: October 27 - November 11, 1997 Grade 66 Silo 4: November 11 - December 5, 1997 Reference: 97-488
IMERYS 051419	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Grade 66 Silo 4: January 5 - 10, 1998 Grade 66 Silo 3: January 12 - 16, 1998 Reference: A98013
IMERYS 051420	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Grade 66 Silo 1: August 12 - 15, 1997 Grade 66 Silo 2: August 15 - September 3, 1997 Reference: 97-357
IMERYS 051421	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Grade 66 Silo 1: December 5 - 11, 1997 Grade 66 Silo 2: December 17 - 31, 1997 Reference: 98002

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051422	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Grade 66 Silo 1: September 22 - 26, 1997 Grade 66 Silo 3: October 1 -7, 1997 Reference: 97-418
IMERYS 051423	Float Feed	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy No asbestiform amphibole minerals were detected Date Milled: April 1995
IMERYS 051423	Grade 36	X-Ray Diffraction	Amphibole Chrysotile	ND* ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy No asbestiform amphibole minerals were detected Date Milled: April 1995
IMERYS 051423	Beta Gamma & Delta	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy No asbestiform amphibole minerals were detected Date Milled: April 1995
IMERYS 051423	Conditioner Slurry	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy No asbestiform amphibole minerals were detected Date Milled: April 1995

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051423	W. W. Product	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: April 1995
IMERYS 051423	W. W. V-710	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: April 1995
IMERYS 051423	W.W. Silo 12 Grade C2+ / V1000	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy No asbestiform amphibole minerals were detected Date Milled: April 12 - 18, 1995
IMERYS 051423	W.W. Silo 8 Grade CO+ (Dense)	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: April 3 - 7, 1995
IMERYS 051423	W.W. Silo 3 Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND* ND	ND: Not detected *: XRD results were corroborated by polarized light microscopy No asbestiform amphibole minerals were detected Date Milled: March 31 - April 12, 1995
IMERYS 051423	W.W. Silo 4 Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND* ND	ND: Not detected *: XRD results were corroborated by polarized light microscopy No asbestiform amphibole minerals were detected Date Milled: April 18 - 21, 1995

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051423	W.W. Silo 7 Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: April 25 - 28, 1995
IMERYS 051424	Float Feed	X-Ray Diffraction	Amphibole	ND*	ND: Not detected *: XRD results were corroborated by polarized light microscopy Date Milled: April 1995 Reference: 95-133
IMERYS 051425	Grade 66 - Silo 5	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: April 24 - May 2, 1995
IMERYS 051425	Grade 66 - Silo 6	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: May 12 - 18, 1995
IMERYS 051426	Grade 66	Polarized Light Microscopy	Serpentine	ND	ND: Not detected Analysis by polarized light microscopy, using dispersion staining technique (1.55 refractive index oil) confirmed the absence of serpentine Grade 66 Composite Silo 1: March 1995 Reference: 95-086
IMERYS 051427	Float Feed	X-Ray Diffraction	Amphibole	ND	ND: Not detected Composite Sample: February 1995 Reference: 95-087

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051428	Float Feed	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: February 1995
IMERYS 051428	Grade 36	X-Ray Diffraction	Amphibole Chrysotile	ND* ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform amphibole minerals were detected Date Milled: February 1995
IMERYS 051428	Beta Gamma & Delta	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform amphibole minerals were detected Date Milled: February 1995
IMERYS 051428	Conditioner Slurry	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: February 1995
IMERYS 051428	W. W. Product	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: February 1995
IMERYS 051428	W. W. V-710	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: February 1995
IMERYS 051428	W. W. Silo 11 Grade CO+	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected

Bates #	Product Tested	Method	Analytes	Results	Comments
					Date Milled: February 16 - March 2, 1995
IMERYS 051428	W. W. Silo 8 Grade CO+ (Dense)	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: February 17 - 23, 1995
IMERYS 051428	W. W. Silo 12 Grade C2+ / V1000	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: February 1 - 11, 1995
IMERYS 051429	Float Feed	X-Ray Diffraction	Amphibole	ND	ND: Not detected *: XRD results were verified by polarized light microscopy Composite Sample: March 1995 Reference: 95-094
IMERYS 051430	Float Feed	X-Ray Diffraction	Amphibole Chrysotile	ND* ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform amphibole minerals were detected Date Milled: March 1995
IMERYS 051430	Grade 36	X-Ray Diffraction	Amphibole Chrysotile	ND* ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform amphibole minerals were detected Date Milled: March 1995

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051430	Beta Gamma & Delta	X-Ray Diffraction	Amphibole Chrysotile	ND* ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform amphibole minerals were detected Date Milled: March 1995
IMERYS 051430	Conditioner Slurry	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform amphibole minerals were detected Date Milled: March 1995
IMERYS 051430	W. W. Product	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: March 1995
IMERYS 051430	W. W. V-710	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: March 1995
IMERYS 051430	W. W. Silo 12 Grade C2+ / V1000	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: March 10 - 17, 1995
IMERYS 051430	W. W. Silo 9 Grade CO+ (Dense)	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform amphibole minerals were detected Date Milled: March 18 - 25, 1995
IMERYS 051430	W. W. Silo 2 Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND* ND	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform amphibole minerals were detected

Bates #	Product Tested	Method	Analytes	Results	Comments
					Date Milled: March 25 - 31, 1995
IMERYS 051435	Float Feed	X-Ray Diffraction	Amphibole	ND	ND: Not detected Date Milled: September 1994 Reference: 94-324
IMERYS 051435	Conditioner Slurry	X-Ray Diffraction	Amphibole	ND	ND: Not detected Date Milled: September 1994 Reference: 94-324
IMERYS 051436	Float Feed	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: September 1994
IMERYS 051436	Grade 36	X-Ray Diffraction	Amphibole Chrysotile	ND* ND	ND: Not detected *: XRD results were verified by polarized light microscopy Date Milled: September 1994
IMERYS 051436	Beta Gamma & Delta	X-Ray Diffraction	Amphibole Chrysotile	ND* ND*	ND: Not detected *: XRD results were verified by polarized light microscopy Date Milled: September 1994
IMERYS 051436	Conditioner Slurry	X-Ray Diffraction	Amphibole Chrysotile	NQ* ND*	ND: Not detected NQ: Mineral concentration <0.1 % - was not quantified *: XRD results were verified by polarized light microscopy The amphibole found in the Conditioner Slurry was determined by polarized light microscopy to be actinolite in the form of cleavage fragments Date Milled: September 1994

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051436	W. W. Product	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: September 1994
IMERYS 051436	W. W. V-710	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: September 1994
IMERYS 051436	Silos 9 & 7 CO+ (HLBD)	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy Date Milled: September 27 - 29 and September 29 - October 5, 1994
IMERYS 051437	Float Feed	X-Ray Diffraction	Amphibole Chrysotile	ND* ND*	ND: Not detected *: XRD results were verified by polarized light microscopy Date Milled: October 1994
IMERYS 051437	Grade 36	X-Ray Diffraction	Amphibole Chrysotile	NQ* ND*	ND: Not detected NQ: Mineral concentration <0.1 % - was not quantified *: XRD results were verified by polarized light microscopy The amphibole found in the Grade 36 was determined by polarized light microscopy to be actinolite in the form of cleavage fragments Date Milled: October 1994
IMERYS 051437	Beta Gamma & Delta	X-Ray Diffraction	Amphibole Chrysotile	ND* ND*	ND: Not detected *: XRD results were verified by polarized light microscopy Date Milled: October 1994
IMERYS 051437	Conditioner Slurry	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy Date Milled: October 1994
IMERYS 051437	W. W. Product	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: October 1994
IMERYS 051437	W. W. V-710	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: October 1994

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051437	W. W. Silo 11 CO+	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: September 15 - 22, 1994
IMERYS 051437	W. W. Silo 9 CO+ (Dense)	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: October 21 - 25, 1994
IMERYS 051438	Float Feed	X-Ray Diffraction	Amphibole	ND*	ND: Not detected *: XRD results were verified by polarized light microscopy Composite Sample: October 1994 Reference: 94-326
IMERYS 051438	Conditioner Slurry	X-Ray Diffraction	Amphibole	ND	ND: Not detected Sample: October 1994 Reference: 94-326
IMERYS 051439	Float Feed	X-Ray Diffraction	Amphibole	ND	ND: Not detected Composite Sample: May 1994 Reference: 94-194
IMERYS 051439	Conditioner Slurry	X-Ray Diffraction	Amphibole	ND*	ND: Not detected *: XRD results were verified by polarized light microscopy Sample: May 1994 Reference: 94-194
IMERYS 051440	Float Feed	X-Ray Diffraction	Amphibole Chrysotile	ND* ND	ND: Not detected *: XRD results were verified by polarized light microscopy Date Milled: July 1994
IMERYS 051440	Grade 36	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy Date Milled: July 1994
IMERYS 051440	Beta & Gamma	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy

Bates #	Product Tested	Method	Analytes	Results	Comments
					Date Milled: July 1994
IMERYS 051440	Conditioner Slurry	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: July 1994
IMERYS 051440	W. W. Product	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: July 1994
IMERYS 051440	W. W. V-710	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: July 1994
IMERYS 051440	Silo 12 C2+ / V1000	X-Ray Diffraction	Amphibole Chrysotile	ND* ND	ND: Not detected *: XRD results were verified by polarized light microscopy Date Milled: July 1994
IMERYS 051440	Silo 9 & 11 CO+	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: 6/30 - 7/15, 7/21 - 7/29/94
IMERYS 051441	Float Feed	X-Ray Diffraction	Amphibole	NQ*	NQ: Mineral concentration <0.1 % - was not quantified *: XRD results were verified by polarized light microscopy Composite Sample: July 1994 Reference: 94-258
IMERYS 051441	Conditioner Slurry	X-Ray Diffraction	Amphibole	NQ*	NQ: Mineral concentration <0.1 % - was not quantified *: XRD results were verified by polarized light microscopy Sample: July 1994 Reference: 94-258
IMERYS 051442	Float Feed	X-Ray Diffraction	Amphibole	NQ*	NQ: Mineral concentration <0.1 % - was not quantified *: XRD results were verified by polarized light microscopy Composite Sample: June 1994

Bates #	Product Tested	Method	Analytes	Results	Comments
					Reference: 94-218
IMERYS 051442	Conditioner Slurry	X-Ray Diffraction	Amphibole	NQ*	NQ: Mineral concentration <0.1 % - was not quantified *: XRD results were verified by polarized light microscopy Sample: June 1994 Reference: 94-218
IMERYS 051443	Float Feed	X-Ray Diffraction	Amphibole	ND*	ND: Not detected *: XRD results were verified by polarized light microscopy Composite Sample: November 1994 Reference: 94-358
IMERYS 051443	Conditioner Slurry	X-Ray Diffraction	Amphibole	ND	ND: Not detected Sample: November 1994 Reference: 94-358
IMERYS 051444	Float Feed	X-Ray Diffraction	Amphibole Chrysotile	ND* ND	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform minerals were detected Date Milled: November 1994
IMERYS 051444	Grade 36	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Date Milled: November 1994

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051444	Beta Gamma & Delta	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform minerals were detected Date Milled: November 1994
IMERYS 051444	Conditioner Slurry	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Date Milled: November 1994
IMERYS 051444	W. W. Product	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform minerals were detected Date Milled: November 1994
IMERYS 051444	W. W. V-710	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Date Milled: November 1994
IMERYS 051444	W. W. Silo 12 Grade C2+ / V1000	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Date Milled: October 10 - 18, 1994
IMERYS 051444	W. W. Silo 12 Grade C2+ / V1000	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Date Milled: November 16 - 17, 1994
IMERYS 051444	W. W. Silo 11 Grade CO+	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected

Bates #	Product Tested	Method	Analytes	Results	Comments
					Date Milled: October 31 - November 4, 1994
IMERYS 051444	W. W. Silo 7 Grade CO+ (Dense)	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Date Milled: November 9 - 16, 1994
IMERYS 051445	Float Feed	X-Ray Diffraction	Amphibole	ND	ND: Not detected Composite Sample: December 1994 Reference: 95-005
IMERYS 051445	Conditioner Slurry	X-Ray Diffraction	Amphibole	ND	ND: Not detected Sample: December 1994 Reference: 95-005
IMERYS 051446	Float Feed	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform minerals were detected Date Milled: December 1994
IMERYS 051446	Grade 36	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform minerals were detected Date Milled: December 1994
IMERYS 051446	Beta Gamma & Delta	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform minerals were detected Date Milled: December 1994

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051446	Conditioner Slurry	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform minerals were detected Date Milled: December 1994
IMERYS 051446	W. W. Product	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform minerals were detected Date Milled: December 1994
IMERYS 051446	W. W. V-710	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Date Milled: December 1994
IMERYS 051446	W. W. Silo 12 Grade C2+ / V1000	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Date Milled: December 6 - 9, 1994
IMERYS 051446	W. W. Silo 6 Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy No asbestiform minerals were detected Date Milled: December 13 - 21, 1994
IMERYS 051446	W. W. Silo 11 Grade CO+	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected No asbestiform minerals were detected Date Milled: December 21 - 30, 1994
IMERYS 051447	Float Feed	X-Ray Diffraction	Amphibole Chrysotile	ND* ND	ND: Not detected *: XRD results were verified by polarized light microscopy

Bates #	Product Tested	Method	Analytes	Results	Comments
					Date Milled: August 1994
IMERYS 051447	Grade 36	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: August 1994
IMERYS 051447	Beta Gamma & Delta	X-Ray Diffraction	Amphibole Chrysotile	ND* ND*	ND: Not detected *: XRD results were verified by polarized light microscopy Date Milled: August 1994
IMERYS 051447	Conditioner Slurry	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: August 1994
IMERYS 051447	W. W. Product	X-Ray Diffraction	Amphibole Chrysotile	ND ND*	ND: Not detected *: XRD results were verified by polarized light microscopy Date Milled: August 1994
IMERYS 051447	W. W. V-710	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: August 1994
IMERYS 051447	W. W. V-1500	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: August 1994
IMERYS 051447	Silos 7 & 11 CO+	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: August 2 - 5 & August 17 - 25, 1994
IMERYS 051447	Silos 9 & 7 CO+ Hi Bulk	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Date Milled: August 8 - 12 & September 6 - 10, 1994
IMERYS 051448	Float Feed	X-Ray Diffraction	Amphibole	NQ*	NQ: Not quantified (<0.1 %) *: XRD results were verified by polarized light microscopy Composite Sample: August 1994 Reference: 94-290

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 051448	Conditioner Slurry	X-Ray Diffraction	Amphibole	NQ	NQ: Not quantified (<0.1 %) Sample: August 1994 Reference: 94-290
IMERYS 058046	Powder	USP 30 - Talc Monograph	Absence of Asbestos A	Negative	Sample ID: 2007-006529-DRPK-001 Sample Description: GUANGXI 1 Date Sampled: 6/1/2007
IMERYS 058048	Powder	USP 30 - Talc Monograph	Absence of Asbestos A	Negative	Sample ID: 2007-006529-DRPK-002 Sample Description: GUANGXI 2 Date Sampled: 6/1/2007
IMERYS 058050	Powder	USP 30 - Talc Monograph	Absence of Asbestos A	Negative	Sample ID: 2007-006529-DRPK-003 Sample Description: GUANGXI 3 Date Sampled: 6/1/2007
IMERYS 058073	Ore	X-Ray Diffraction: CTFA J4-1 X-Ray Diffraction: CTFA J6-1 Transmission Electron Microscopy: TM 7024	Initial Ore Results - After Grinding (1B) Fibrous Amphibole Results - After Grinding (1C) Asbestos	ND ND	ND: None detected Composite Sample: Guangxi #2 (MV Makali) Reference: A07137-1
IMERYS 058073	Ore	X-Ray Diffraction: CTFA J4-1 X-Ray Diffraction: CTFA J6-1 Transmission Electron Microscopy: TM 7024	Initial Ore Results - After Grinding (1B) Fibrous Amphibole Results - After Grinding (1C) Asbestos	ND ND	ND: None detected Composite Sample: Guangxi #2A (MV Makali) Reference: A07137-2
IMERYS 058075	Ore	ASTM Draft Method "Standard Test Method for Indirect Analysis of Talc by Transmission Electron Microscopy for Asbestos Mass Concentration" Modification of ASTM Method D 5756-02	Amphibole Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration Chrysotile Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration	0 0 BDL 0 0 BDL	BDL: Below detection limit Sample of Guangxi #1 (M.V. Makali): RM, ACM, AFG Composite: July 15, 2007 Reference: A07140-1

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 058221	Talc	USP 32 - NF 27	Absence of Asbestos A	Negative	Sample ID: 2010-DRPK-000997-001 Representing: M.V. Blunenau Grind Date: 10/06/09
IMERYS 058222	Ore	X-Ray Diffraction: CTFA J4-1 X-Ray Diffraction: CTFA J6-1 Transmission Electron Microscopy: TM 7024	<u>Initial Ore Results - After Grinding (1B)</u> Fibrous Amphibole <u>Results - After Grinding (1C)</u> Asbestos	ND ND	ND: None detected Composite Sample: Guangxi #2 Composite MV Blunenau Reference: A10044-1
IMERYS 090928	Grade 66	Johnson & Johnson Test No. TM7024 ("Analysis of Powdered Talc for Asbestiform Minerals by Transmission Electron Microscopy," REV: 08/21/95)	Total Asbestos	N/D	N/D: None detected Grade 66 Composite: 4th Quarter 1999 Dates Milled: November 18 - 21, 1999; December 1 - 6, 1999; December 17 - 22, 1999; January 5 - 8, 2000 Reference: A00003
IMERYS 102508	Cosmetic Talc	X-Ray Diffraction Polarized Light Microscopy	Serpentine Chrysotile	Trace ND	ND: Not detected Composite Sample West Windsor Silo 2 Batch: 1940-1945
IMERYS 102508	Cosmetic Talc	X-Ray Diffraction Polarized Light Microscopy	Serpentine Chrysotile	0.1 % ND	ND: Not detected Composite Sample West Windsor Silo 3 Batch: 1964 - 1968
IMERYS 113435	Ore	X-Ray Diffraction: CTFA J4-1 X-Ray Diffraction: CTFA J6-1 Transmission Electron Microscopy: TM 7024	<u>Initial Ore Results - After Grinding (1B)</u> Fibrous Amphibole <u>Results - After Grinding (1C)</u> Asbestos	ND ND	ND: None detected (<2 %) Composite Sample: Guangxi #2A October 2003 Shipment (Tian Song Feng MV) Ore Shipment ID: A03491, A03613-2, A04042-1

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 113435	Ore	X-Ray Diffraction: CTFA J4-1 X-Ray Diffraction: CTFA J6-1	Initial Ore Results - After Grinding (1B) Fibrous Amphibole	ND	ND: None detected (<2 %) Composite Sample: Guangxi #2A February 2004 Shipment (MV Tequi) Reference: A04111-1
IMERYS 113570	Talc	USP 31 - NF 26	Absence of Asbestos A	Negative	Sample ID: 2009-DRPK-000398-001 Representing: Guangxi 1: January 2009
IMERYS 113570	Talc	USP 31 - NF 26	Absence of Asbestos A	Negative	Sample ID: 2009-DRPK-000398-002 Representing: Guangxi 1: January 2009
IMERYS 113582	Ore	X-Ray Diffraction: CTFA J4-1 X-Ray Diffraction: CTFA J6-1 Transmission Electron Microscopy: TM 7024	Initial Ore Results - After Grinding (1B) Fibrous Amphibole Results - After Grinding (1C) Asbestos	ND ND	ND: None detected Composite Sample: Guangxi #2 (MV Anna Smile): January 2008 Ore Shipment ID: A09020-1
IMERYS 113584	Ore	ASTM Draft Method "Standard Test Method for Indirect Analysis of Talc by Transmission Electron Microscopy for Asbestos Mass Concentration" Modification of ASTM Method D 5756	Amphibole Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration Chrysotile Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration	0 0 BDL 0 0 BDL	BDL: Below detection limit Composite Sample of Guangxi #1 (M.V. Anna Smile): January 23, 2009 Reference: A09018-1
IMERYS 113607 IMERYS 113608	Talc	USP 31 - NF 26	Absence of Asbestos A	Negative	Sample ID: 2009-DRPK-011976-001 Representing: Guangxi 1: October 2009
IMERYS 113616	Ore	X-Ray Diffraction: CTFA J4-1 X-Ray Diffraction: CTFA J6-1 Transmission Electron Microscopy: TM 7024	Initial Ore Results - After Grinding (1B) Fibrous Amphibole Results - After Grinding (1C) Asbestos	ND ND	ND: None detected Composite Sample: Guangxi #2 (MV Beilun Dolphin): October 30, 2009 Ore Shipment ID: A09481-1
IMERYS 198887	Talc	Not Clear	Asbestos	NIL	Commodity: Guangxi Talc Lumps No. 2 S/C No: 2012L-021 Date: December 25, 2012

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 209012	Grade 66	Johnson & Johnson Test No. TM7024 ("Analysis of Powdered Talc for Asbestiform Minerals by Transmission Electron Microscopy," REV: 08/21/95)	Total Asbestos	N/D	N/D: None detected Grade 66 Composite: 4th Quarter 1999 Dates Milled: October 21 - 27, 1998; November 25 - December 8, 1998; December 8 - December 31, 1998; December 31, 1998 - January 7, 1999 Reference: A98444
IMERYS 209013	Grade 66	Not Clear	Amphibole	ND	ND: Not detected Grade 66 Silos 1 & 2 Dates Milled: February 22 - 25, 1999; February 25 - 28, 1999 No asbestiform minerals were detected Reference: A99111
IMERYS 209014	Grade 66	Not Clear	Amphibole	ND	ND: Not detected Grade 66 Silos 3 & 4 Dates Milled: January 30 - February 3, 1999; February 15 - 17, 1999 No asbestiform minerals were detected Reference: A99061
IMERYS 209015	Grade 66	Not Clear	Amphibole	ND	ND: Not detected Grade 66 Silos 1 & 2 Dates Milled: December 8 - 31, 1998; December 31, 1998 - January 7, 1999 No asbestiform minerals were detected Reference: A99014

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 209016	Grade 66	Johnson & Johnson Test No. TM7024 ("Analysis of Powdered Talc for Asbestiform Minerals by Transmission Electron Microscopy," REV: 08/21/95)	Total Asbestos	N/D	N/D: None detected Grade 66 Composite: 3rd Quarter 1998 Dates Milled: July 18, - 22, 1998; July 22 - 29, 1998; August 5 - 9, 1998; August 27 - September 2, 1998; September 11 - 18, 1998 Reference: A98354
IMERYS 209017	Grade 66	Johnson & Johnson Test No. TM7024 ("Analysis of Powdered Talc for Asbestiform Minerals by Transmission Electron Microscopy," REV: 08/21/95)	Total Asbestos	N/D	N/D: None detected Grade 66 Composite: 2nd Quarter 1998 Dates Milled: May 1 -15, 1998; May 15 - 22, 1998; June 23 - July 18, 1998 Reference: A98288
IMERYS 209018	Grade 66	Johnson & Johnson Test No. TM7024 ("Analysis of Powdered Talc for Asbestiform Minerals by Transmission Electron Microscopy," REV: 08/21/95)	Total Asbestos	N/D	N/D: None detected Grade 66 Composite: 1st Quarter 1998 Dates Milled: January 5 - 10, 1998; January 12 - 16, 1998; January 22 - 26, 1998; February 11 - 15, 1998; February 15 - 19, 1998; February 25 - March 2, 1998; March 2 - 5, 1998; March 25 - 29, 1998; March 29 - April 2, 1998; April 2 - 8, 1998 Reference: A98014
IMERYS 209019	Grade 66	Johnson & Johnson Test No. TM7024 ("Analysis of Powdered Talc for Asbestiform Minerals by Transmission Electron Microscopy," REV: 08/21/95)	Total Asbestos	N/D	N/D: None detected Grade 66 Composite: 4th Quarter 1997 Dates Milled: October 9 - 10, 1997; October 24 - 27, 1997; October 27 - November 11, 1997; November 11 - December 5, 1997; December 5 - 11, 1997; December 17 - 31, 1997 Reference: 97-448

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 209020	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Grade 66 Silos 1 & 2 Date Milled: January 22 - 26, 1998; February 11 - 15, 1998 Reference: A98047
IMERYS 209021	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Grade 66 Silos 3 & 4 Date Milled: October 27 - November 11, 1997; November 11 - December 5, 1997 Reference: 97-488
IMERYS 209022	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Grade 66 Silos 4 & 3 Date Milled: January 5 - 10, 1998; January 12 - 16, 1998 Reference: A98013
IMERYS 209023	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Grade 66 Silos 1 & 2 Date Milled: August 12 - 15, 1997; August 15 - September 3, 1997 Reference: 97-357
IMERYS 209024	Grade 66	X-Ray Diffraction	Amphibole Chrysotile	ND ND	ND: Not detected Grade 66 Silos 1 & 2 Date Milled: December 5 - 11, 1997; December 17 - 31, 1997 Reference: 98002

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210700	Vermont 66	X-Ray Diffraction	Amphibole (Including Tremolite) Serpentine (Including Chrysotile)	ND ND	ND: Not detected Composite Sample: Vermont 66 Talc Dates: May 10 -15, 1976; May 17 - 22, 1976; May 24 - 29, 1976; June 1 - 4, 1976; June 7 - 11, 1976; June 14 - 18, 1976; June 21 - 25, 1976; June 28 - July 2, 1976; July 6 - 10, 1976; July 12 - 16, 1976; July 19 - 23, 1976; July 26 - 30, 1976; August 2 - 6, 1976; August 9 - 13, 1976; August 16 - 20, 1976 Reference: 0503.01 Data from a letter - not a lab report
IMERYS 210701	Vermont 66	X-Ray Diffraction	Amphibole Serpentine	ND ND	ND: Not detected Composite Sample: Vermont 66 Talc Dates: August 23 - 29, 1976 August 30 - September 3, 1976; September 7 - 10, 1976; September 20 - 24, 1976 Reference: 0503.01
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: W-GI (Rolled talc, organic fiber and talc ribbons) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: BI-GI (Silicates and talc ribbons) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: BI-WI (Blocky talc, crystalline square particles) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Low	Low: 1 -3 fibers found Sample No.: FI-WI (Large particles, 1 amphibole, 1 fibrous antigorite, silicates, and rolled talc) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: Y-GI (Some organic material, fine crystalline particles about 500 Å in size and silicates) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: W-HC (Blocky talc and organic material) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: V-HC (Organic material) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: Z-GT No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: Y-HC (Silicates) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: DI-HC (Blocky talc, 2 silicate fibers) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: GI-HC No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Low	Low: 1 -3 fibers found Sample No.: X-HC (2 amphiboles, 1 talc hard) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Medium	Medium: 4 - 8 fibers found Sample No.: FI-HC (2 bundles of amphiboles, 2 single amphibole fibers) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: V-WI (2 silicates) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Low	Low: 1 -3 fibers found Sample No.: V-GI (2 amphibole and 1 amphibole-like fiber without diffraction pattern 2 talc ribbons, fine particulate contamination and organic crud) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Low	Low: 1 -3 fibers found Sample No.: EI-HC No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Low	Low: 1 -3 fibers found Sample No.: GI-WI (Blocky talc, talc fibers, silicates, 1 amphibole) No chrysotile (serpentine asbestos) found

Bates #	Product Tested	Method	Analytes	Results	Comments
					From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: CI-HC (Bacteria, silicates, blocky talc and organic fibrils) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: CI-HC (Bacteria, silicates, blocky talc and organic fibrils) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: DI-GI (Blocky talc, organic material, rolled talc and silicates) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Low	Low: 1 -3 fibers found Sample No.: CI-GI (Silicates, talc ribbon, fibrous talc, blocky talc, organic fibers and 2 bundles of amphibole) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: U-GI (Organic material, blocky talc, and silicates) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: HI-HC No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: HI-WI No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Low	Low: 1 -3 fibers found Sample No.: BI-HC (Rolled talc fibers, blocky talc and 2 amphibole bundles) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: EI-GI No chrysotile (serpentine asbestos) found From letter dated July 1, 1975

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: AI-HC (Silicates) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: EI-WI (Blocky talc) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Low	Low: 1 -3 fibers found Sample No.: Z-HC (Small square particulate matter about 1,000 Å, 3 bundles of amphibole) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: DI-WI (1 amphibole, fine particles, fibrous talc and blocky talc) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: HI-WI No chrysotile (serpentine asbestos) found From letter dated July 1, 1975

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: BI-HC (Rolled talc fibers, blocky talc and 2 amphibole bundles) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Low	Low: 1 -3 fibers found Sample No.: EI-GI No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: Y-GI (Some organic material, fine crystalline particles about 500 Å in size and silicates) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: U-HC No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: W-GI (Rolled talc, organic fiber and talc ribbons) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: Z-GI No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Low	Low: 1 -3 fibers found Sample No.: EI-WI (Blocky talc) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: GI-HC No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: Y-HC (Silicates) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: DI-GI (Blocky talc, organic material, rolled talc and silicates) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: FI-WI (Large particles, 1 amphibole, 1 fibrous antigorite, silicates and rolled talc) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: W-HC (Blocky talc and organic material) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: V-WI (Silicates) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: U-GI (Organic material, blocky talc and silicates) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Low	Low: 1 -3 fibers found Sample No.: Z-HC (Small square particulate matter about 1,000 Å, 3 bundles of amphibole No chrysotile (serpentine asbestos) found From letter dated July 1, 1975

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: X-HC (2 amphiboles, 1 talc hard) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: CI-HC (Bacteria, silicates, blocky talc and organic fibers) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: DI-HC (Blocky talc, 2 silicate fibers) No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Low	Low: 1 -3 fibers found Sample No.: D-HC 7/22 No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: D-WI 7/15 No chrysotile (serpentine asbestos) found From letter dated July 1, 1975

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: D-GI 7/15 No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: F-HC 9/3 No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: H-GI 9/16 No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	0	0: None found Sample No.: I-WI No chrysotile (serpentine asbestos) found From letter dated July 1, 1975
IMERYS 210702 IMERYS 210703 IMERYS 210704 IMERYS 210705 IMERYS 210706	Talc	Electron Microscopy and Selected Area Electron Diffraction	Confirmed Asbestos, Visual	Low	Low: 1 -3 fibers found Sample No.: P-GI No chrysotile (serpentine asbestos) found From letter dated July 1, 1975

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210707 IMERYS 210708	Grade 36 Columbia Grades X-42 - C-83 West Windsor Float Feed X-42 - C-83 Black Bear Core Sample Clifton Float	Transmission Electron Microscopy	Asbestiform Minerals	ND	ND: Not detected # Samples: 20 # Detections: 0 Date Range: February 1984 - September 1984
IMERYS 210709 IMERYS 210710	Hammondsville Ore TC-100 Columbia Mill (Industrial)	Transmission Electron Microscopy	Asbestos Minerals	ND*	ND: Not detected * Samples were found to contain no asbestos minerals and very small amounts of antigorite were found in a few samples # Samples: 21 # Detections: 0 Date Range: January 1983 - July 1983
IMERYS 210724	Talc	Transmission Electron Microscope and Selected Area Electron Diffraction	Asbestos Minerals	ND*	ND: Not detected * Not asbestos minerals were found in any of the samples. A fibrous clay (sepiolite) was found in all samples in low percentages. # Samples: 7 # Detections: 0 Date: June 10, 1983
IMERYS 210725 IMERYS 210726	Hammondsville Ore TC-100 Columbia Mill (Industrial)	Transmission Electron Microscopy	Asbestos Minerals	ND	ND: Not detected # Samples: 33 # Detections: 0 Date Range: April 1982 - January 1983
IMERYS 210729 IMERYS 210730	Talc	X-Ray Diffraction and Transmission Electron Microscope	Serpentine Amphibole	Detected ND*	ND: Not detected * A small amount of antigorite was found. Talc sample from Vermont, submitted with letter of January 25, 1983

Bates #	Product Tested	Method	Analytes	Results	Comments
					Reference: 4055
IMERYS 210738 IMERYS 210739	Hammondsville Ore TC-100 Columbia Mill (Industrial)	Transmission Electron Microscope	Asbestos Minerals	ND	ND: Not detected # Samples: 27 # Detections: 0 Date Range: August 1981 - April 1982
IMERYS 210743	Hammondsville Ore TC-100 Columbia Mill (Industrial)	Transmission Electron Microscope	Asbestos Minerals	ND	ND: Not detected # Samples: 19 # Detections: 0 Date Range: July 1980 - January 1981 Reference: 4055
IMERYS 210758 IMERYS 210759	Talc	Transmission Electron Microscope	Asbestos Minerals	ND (18 Samples) Chrysotile, 1 fiber (1 Sample)	ND: Not detected # Samples: 19 # Detections: 1 Results letter dated: September 8, 1980 Reference: 4055
IMERYS 210788 IMERYS 210789	Talc	Transmission Electron Microscope and Light Microscopy	Asbestiform and Potentially Asbestiform Minerals	ND	ND: Not detected # Samples: 9 # Detections: 0 Results letter dated: March 27, 1980

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210792 IMERYS 210793 IMERYS 210794	Hammondsville Ore TC-100 Columbia Mill (Industrial)	Transmission Electron Microscope	Asbestos Minerals	ND	ND: Not detected # Samples: 18 # Detections: 0 Date Range: June 1979 - November 1979
IMERYS 210795 IMERYS 210796	Hammondsville Ore TC-100 Columbia Mill (Industrial)	Transmission Electron Microscope	Asbestos Minerals	ND	ND: Not detected # Samples: 18 # Detections: 0 Date Range: January 1979 - June 1979
IMERYS 210797	Talc	Transmission Electron Microscope and Selected Area Electron Diffraction	Asbestiform Minerals	ND	ND: Not detected # Samples: 2 # Detections: 0 Results letter dated: March 5, 1979
IMERYS 210800	Talc	JEM-200 Transmission Electron Microscope	Chrysotile	12 fibers*	* 12 fibers observed in 10 squares of the support grid. *"This extrapolates to a total of 650x10³ fibers on the entire filter. 10 of the (diam. approx. 0.035 µm) 12 fibers observed were unit fibrils of chrysotile about 0.5-0.6 µm long. The other two were only slightly larger with diameters of about 0.06-0.07 µm and lengths of about 2 µm."* Filter sample submitted August 25, 1978
IMERYS 210801	Talc	Not Specified	Asbestiform Minerals	ND (36 Samples) 1 Chrysotile Fiber of Approximately 0.5 µm in length (2 Samples)	# Samples: 38 # Detections: 2 Date: 1978 Results letter notes that both fibers could be contamination from outside sources.

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 210805 IMERYS 210806	Talc	Transmission Electron Microscope	Asbestiform Minerals	ND	ND: Not detected # Samples: 45 # Detections: 2 Date Range: May 1977 - October 1977
IMERYS 210809	Talc	Transmission Electron Microscope and Selected Area Electron Diffraction; X-Ray Diffraction	Asbestiform Minerals	ND	ND: Not detected # Samples: 1 # Detections: 0 Date: November 1, 1977
IMERYS 210810 IMERYS 210811 IMERYS 210812	Talc	Transmission Electron Microscope	Serpentine Chrysotile	Present (Detected in 4 Samples) Present (Detected in 1 Sample) *	# Samples: 40 # Detections: 5 Date Range: 1976 - 1977 * Detected at a level in the order of ppt. Sample was re-examined three more times and no chrysotile was found. In addition, another ore sample from the same location was also round and examined and again found no chrysotile
IMERYS 210810 IMERYS 210811 IMERYS 210812	Talc	Transmission Electron Microscope	Asbestos Minerals	ND	ND: Not detected # Samples: 35 # Detections: 0 Date Range: October 1976 - May 1977
IMERYS 210824 IMERYS 210825	Industrial Grade Talc	Transmission Electron Microscope and Selected Area Electron Diffraction	Asbestiform Minerals Chrysotile	ND 1 Fiber (1 Sample) * 5 Particles (1 Sample)	ND: Not detected # Samples: 35 # Detections: 2 Date Range: August 1975 - February 1976

Bates #	Product Tested	Method	Analytes	Results	Comments
					*Believed to be background contamination
IMERYS 210826 IMERYS 210827	Talc	SAED and Electron Microscopy	Asbestiform Minerals	ND	ND: Not detected # Samples: 3 # Detections: 0 Date Range: September 1975 - January 1976 Project No.: 0503.01
IMERYS 210839	Personal Air Filter Samples	Transmission Electron Microscope and Selected Area Electron Diffraction	Asbestiform Minerals	ND	ND: Not detected # Samples: 4 # Detections: 0 Reference: MA 4055
IMERYS 210852 IMERYS 210853 IMERYS 210854	Talc	Not Specified	Asbestiform Materials	ND	ND: Not detected # Samples: 29 # Detections: 0 Results letter dated: December 31, 1974
IMERYS 213431	Cosmetic Talc	X-Ray Diffraction and PLM	Serpentine Chrysotile	0.9 % ND	ND: Not detected Composite Grade 320V Silo 5: 11/30 - 12/1/99 Reference: A99543-1

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 213431	Cosmetic Talc	X-Ray Diffraction and PLM	Serpentine Chrysotile	0.4 % ND	ND: Not detected Composite Grade 66 Silo 2: 12/1 - 6/99 Reference: A99543-2
IMERYS 418940	Vermont Classified XP Talc	X-Ray Diffraction	Serpentine	3.60%	Date: January 8, 2009 Reference: A08669-1
IMERYS 427237	Rainbow Mine Whole Rock	X-Ray Diffraction	Amphibole Serpentine	ND ND	ND: Not detected Date: September 6, 1990 # Samples: 5 All samples from 1270 Level
IMERYS 427237	Rainbow Mine Sludge	X-Ray Diffraction	Amphibole Serpentine	ND ND	ND: Not detected Date: August 29, 1990 # Samples: 9
IMERYS 427238	Rainbow Mine Core	X-Ray Diffraction	Amphibole Serpentine	ND ND	ND: Not detected Date: September 19, 1990 # Samples: 6
IMERYS 427238	Rainbow Mine Core	X-Ray Diffraction	Amphibole Serpentine	ND ND	ND: Not detected Date: September 20, 1990 # Samples: 7
IMERYS 427239	Argonaut Mine Whole Rock	X-Ray Diffraction	Amphibole Serpentine	ND ND	ND: Not detected Date: November 16, 1990 # Samples: 15
IMERYS 477880	Grade 66	Johnson & Johnson Test No. TM7024 ("Analysis of Powdered Talc for Asbestiform Minerals by Transmission Electron Microscopy," REV: 08/21/95)	Total Asbestos	N/D	N/D: None detected Composite 1st quarter sample: 1999 Reference: A99062

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 499264	Argonaut Drilling Sample	Polarized Light Microscopy	Tremolite	4%	Results letter date: March 31, 2003 Reference: A02703
IMERYS 469484	Argonaut South MacKenzie Lease	Denver X-Ray Diffraction	Serpentine	0.1 - 4.3 %	# Samples: 8 # Detections: 3 Date Range: May 24-25, 2001
IMERYS 469485	Argonaut South MacKenzie Lease	Denver X-Ray Diffraction	Serpentine	0.8 - 3.3 %	# Samples: 10 # Detections: 2 Date: May 25, 2001
IMERYS 469486	Argonaut South	Denver X-Ray Diffraction	Serpentine	0.1 - 0.6 %	# Samples: 12 # Detections: 3 Date: May 26, 2001
IMERYS 469487	Argonaut South	Denver X-Ray Diffraction	Serpentine	0.10%	# Samples: 17 # Detections: 1 Date: May 27, 2001
IMERYS 469488	Argonaut South	Denver X-Ray Diffraction	Serpentine	0.1 - 0.4 %	# Samples: 15 # Detections: 9 Date: May 28, 2001
IMERYS 469489	Argonaut South	Denver X-Ray Diffraction	Serpentine	0.1 - 0.2 %	# Samples: 19 # Detections: 3 Date: May 28-29, 2001
IMERYS 469491	Argonaut South	Denver X-Ray Diffraction	Serpentine	0.2 - 1.0 %	# Samples: 12 # Detections: 4 Date: May 29-30, 2001

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 469492	Argonaut South	Denver X-Ray Diffraction	Serpentine	0.20%	# Samples: 9 # Detections: 3 Date: May 30-31, 2001
IMERYS 469493	Argonaut South	Denver X-Ray Diffraction	Serpentine	0.10%	# Samples: 3 # Detections: 1 Date: May 31, 2001
IMERYS 469494	Argonaut South	Denver X-Ray Diffraction	Serpentine	0.1 - 0.2 %	# Samples: 17 # Detections: 5 Date: June 1-2, 2001
IMERYS-MDL-AB_0005572	Troy Mine Core	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected # Samples: 2 (Floated Product and Feed) 70'-95'
IMERYS-MDL-AB_0005572	Troy Mine Core	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected # Samples: 2 (Floated Product and Feed) 170'-195'
IMERYS-MDL-AB_0005572	Troy Mine Core	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected # Samples: 2 (Floated Product and Feed) 20'-40'
IMERYS-MDL-AB_0005572	Troy Mine Core	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected # Samples: 2 (Floated Product and Feed) 135'-160'
IMERYS-MDL-AB_0005572	Troy Mine Core	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected # Samples: 2 (Floated Product and Feed) 75'-100'

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS-MDL-AB_0005572	Troy Mine Core	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected # Samples: 2 (Floated Product and Feed) 300'-312'
IMERYS-MDL-AB_0005572	Troy Mine Core	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected # Samples: 2 (Floated Product and Feed) 225'-250'
IMERYS-MDL-AB_0005573	Troy Mine Core	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected # Samples: 2 (Floated Product and Feed) 38'-50'
IMERYS-MDL-AB_0005573	Troy Mine Core	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected # Samples: 2 (Floated Product and Feed) 75'-100'
IMERYS-MDL-AB_0005573	Troy Mine Core	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected # Samples: 2 (Floated Product and Feed) 25'-50'
IMERYS-MDL-AB_0005573	Troy Mine Core	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected # Samples: 1 (Rougher Tails composite)
IMERYS-MDL-AB_0005574	Johnson Mill C-0	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 1/29/1988
IMERYS-MDL-AB_0005574	Johnson Mill 1,000 and C-2	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: February 1988
IMERYS-MDL-AB_0005574	Johnson Mill V-6	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 2/5/1988

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS-MDL-AB_0005574	Johnson Mill USPS	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: December 1988
IMERYS-MDL-AB_0005574	Johnson Mill Vertal 200	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: November 1987
IMERYS-MDL-AB_0005574	Johnson Mill 300	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 2/1/1988
IMERYS-MDL-AB_0005574	Johnson Mill 300	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 11/16-30/1989
IMERYS-MDL-AB_0005574	Johnson Mill 700	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 2/2/1988
IMERYS-MDL-AB_0005574	Johnson Mill 700	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 11/16-30/1987
IMERYS-MDL-AB_0005574	Johnson Mill 1,500	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 2/2/1988
IMERYS-MDL-AB_0005574	Johnson Mill C-1	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 2/2/1988
IMERYS-MDL-AB_0005574	Johnson Mill USPS	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 2/7/1988
IMERYS-MDL-AB_0005575	Johnson Mill, Troy Mine Ore Flotation Feed	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 3/29/1988
IMERYS-MDL-AB_0005575	Johnson Mill, Troy Mine Ore C-0	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 3/29/1988
IMERYS-MDL-AB_0005575	Johnson Mill, Troy Mine Ore JS-30	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 3/29/1988
IMERYS-MDL-AB_0005575	Johnson Mill, Troy Mine Ore 300	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 3/29/1988
IMERYS-MDL-AB_0005575	Johnson Mill, Troy Mine Ore 700	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected Date Milled: 3/29/1988

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS-MDL-AB_0005576	Chester Mill, Feed from Hamm Open Pit	Not Specified	Amphibole Serpentine	0.02 - 0.17 % TR	TR: Trace # Samples: 3 Date Milled: 1/5/1987, 3/29/1988 (2 Samples)
IMERYS-MDL-AB_0005577	Vermont Talc Ore from E. Reade	Not Specified	Amphibole Serpentine	ND - ~10 % (1 Detection) ND - ~1 % (2 Detections)	ND: Not detected # Samples: 6
IMERYS-MDL-AB_0005578	Vermont Talc Flotation Concentrates	Not Specified	Amphibole Serpentine	ND ND	ND: Not detected # Samples: 11 Processed at Alpine, Alabama
IMERYS 048393 IMERYS 048394 IMERYS 048395 IMERYS 048396 IMERYS 048397 IMERYS 048398 IMERYS 048399	Hamm Mine Ore	Not Specified	Amphibole Serpentine	ND - >1 0 - V. High	ND: Not detected Hamm Mine Assay Data: October 1992 # Samples: 375 # Amphibole Detections: 53 # Serpentine Detections: 94 Units not specified; V. High not defined
IMERYS-5	Argonaut Product Composites, Ludlow Fine	Not Specified	<u>Amphibole</u> Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration <u>Chrysotile</u> Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration	 0 0 BDL 0 1 - 2 BDL	BDL: Below the detection limit # Samples: 43 # Detections: 8 Dates: Monthly January 2002 - June 2005, 3rd Quarter 2005
IMERYS-5	Argonaut Product Composites, Ludlow Coarse	Not Specified	<u>Amphibole</u> Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration <u>Chrysotile</u> Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration	 0 0 BDL 0 1 BDL	BDL: Below the detection limit # Samples: 43 # Detections: 10 Dates: Monthly January 2002 - June 2005, 3rd Quarter 2005

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS-5	Argonaut Product Composites, Grade 96	Not Specified	<u>Amphibole</u> Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration <u>Chrysotile</u> Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration	0 0 BDL 0 1 BDL	BDL: Below the detection limit # Samples: 7 # Detections: 3 1st, 2nd, 3rd, 4th Quarter 2002; 1st, 2nd, 3rd Quarters 2003
IMERYS-5	Argonaut Product Composites, Float Feed	Not Specified	<u>Amphibole</u> Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration <u>Chrysotile</u> Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration	0 0 BDL 0 1 BDL	BDL: Below the detection limit # Samples: 27 # Detections: 3 Dates: March 2001, Monthly August 2001 - September 2003
IMERYS-5	Argonaut Product Composites, Grade 66	Not Specified	<u>Amphibole</u> Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration <u>Chrysotile</u> Number of Structures >5 µm Number of Structures ≤ 5 µm Asbestos Concentration	0 0 BDL 0 0 BDL	BDL: Below the detection limit # Samples: 4 # Detections: 0 2nd, 3rd, 4th Quarter 2001; December 2001 (Silo 4)
J&J-28	Shower to Shower Body Powder	Not Specified	Tremolite Chrysotile	ND 5% Wt	ND: Not Detected Results letter date: August 3, 1972 (Professor Seymour Lewin of NYU)
J&J-34	Baby Powder	X-Ray Diffraction, Light Microscopy, TEM, Electron Diffraction	Tremolite Chrysotile	A Few Individual Crystals ND	ND: Not detected Results letter date: October 27, 1972 Batch Numbers: 108T, 100T
J&J-0083355	Ultrawet D.S. Trial Period Ore Ultrawet D.S. Trial Period Product	Not Specified	Amphibole	3000 ppm (Ore) 100-200 ppm (Product)	Reagent Trial Process Samples at Windsor Minerals
J&J-0083355	N-Butanol Trial Period Ore N-Butanol Trial Period Product	Not Specified	Amphibole	3000 ppm (Ore) 100-200 ppm (Product)	Reagent Trial Process Samples at Windsor Minerals

Bates #	Product Tested	Method	Analytes	Results	Comments
J&J-0083355	N-Butanol Citric Acid Trial Period Ore N-Butanol Citric Acid Trial Period Product	Not Specified	Amphibole	3000 ppm (Ore) 100-200 ppm (Product)	Reagent Trial Process Samples at Windsor Minerals
J&J-0083361	Grade 66-U-Ore Grade 66 -U-Product	Electron Microscopy	Asbestiform Fiber Count	0 1 (Per E.M. Grid)	Time period using ultrawet D.S. "Probably chrysotile"
J&J-0083361	Grade 66-A-Ore Grade 66 -A-Product	Electron Microscopy	Asbestiform Fiber Count	1 (Per E.M. Grid) 0	Time period using n-butanol "Probably chrysotile"
J&J-0083361	Grade 66-AC-Ore Grade 66 -AC-Product	Electron Microscopy	Asbestiform Fiber Count	8 (Per E.M. Grid) 1	Time period using n-butanol-citric acid "Chrysotile"
J&J-58	Talc Ore	Not Specified	Actinolite Found Actinolite Nominal	4.6 mg --	Study: Windsor Minerals Analysis of Talc Products and Ores
J&J-58	Talc Ore Talc Product	Not Specified	Actinolite	2,300 ppm 170 ppm	Study: Windsor Minerals Analysis of Talc Products and Ores
J&J-65	Talc Ore Core	X-Ray Diffraction	Asbestiform Minerals	ND - 7 (Chrysotile) *Units Not Specified*	Examination of Talc Samples: Argonaut Ore Body ND: Not detected # Samples: 41 # Detections: 1 Report date: April 24, 1974
J&J-65	Talc Ore Core	Electron Microscopy	Chrysotile Amphibole	ND - 0.007 % ND - 0.001 %	Examination of Talc Samples: Argonaut Ore Body ND: Not detected # Samples: 41 # Detections: 15 (Chrysotile), 4 (Amphibole) Report date: April 24, 1974

Bates #	Product Tested	Method	Analytes	Results	Comments
J&J-74	Windsor Minerals Talc	Not Specified	Asbestiform Materials	ND - 0.006 % (Chrysotile)	ND: Not Detected # Samples: 11 # Detections: 1 Report date: October 10, 1974
J&J-169	Windsor Minerals Talc	Transmission Electron Microscopy	Asbestos Minerals	<0.5 % (Chrysotile)	"Several fibers were found so it probably is not a contaminant of the sample" # Samples: 1 Results letter date: November 6, 1980
J&J-100	Talc	X-Ray Diffraction and Microscopies Studies	Asbestos Minerals	Slight Trace (<0.1 %) Tremolite-Actinolite (2 Samples) Slight Trace (<0.1 %) Anthophyllite (1 Sample)	# Samples: 5 Results letter date: February 26, 1973 Reference: C10704
J&J-179	Air Filters	Transmission Electron Microscopy	Asbestos Fibers	<6.6 x 10^3 - 6.0 x 10^4 Fibers/Filter (Chrysotile)	# Samples 4 # Detections: 4 Results letter date: November 2, 1984
J&J-182	Windsor Minerals Talc	Transmission Electron Microscopy	Asbestos Fibers	0.0003 % - 0.0055 % Wt (Chrysotile)	# Samples 3 # Detections: 3 Results letter date: April 29, 1986 Reference: ME-2275
J&J-182	Windsor Minerals Talc	Transmission Electron Microscopy	Asbestos	BDL	BDL: Below detection limit # Samples 19 # Detections: 0 Results letter date: January 28, 1987 Reference: ME-3241

Bates #	Product Tested	Method	Analytes	Results	Comments
J&J-185	Windsor Minerals Raw Talc	Dispersion Staining with Acid Wash	Amphibole Particles	0 - 75 Amphiboles/Slide 150 - 700 Amphiboles/Half Slide	# Samples 27 # Detections: 23 Date Range: March 5-20, 1987 "No fibrous forms were observed"
J&J-185	Windsor Minerals Talc (Loose Grain Counts)	Microscopic Examination	Tremolite Mica, Chlorite, Serpentine	ND - TR (3 Samples) ND - TR (1 Sample)	TR: Trace ND: Not detected # Samples 8 # Detections: 4 Date Range: February 12, February 20, March 2, 1987 In Floatation Feed and Concentrates of Windsor Talc Concentrate
J&J-185	Windsor Minerals Talc (Loose Grain Counts)	Microscopic Examination	Tremolite Mica, Chlorite, Serpentine	ND - 6 % (6 Samples) ND - 6 % (1 Sample)	ND: Not detected # Samples 7 # Detections: 7 Date Range: February 12, February 20, March 2, 1987 In Roller Mill Feed and Products of High Intensity Magneito, Static Belt and Archimedes Spiral Separation of Windsor Talc
J&J-177	Personal Air Filter	Optical and Electron Microscope	Unknown Asbestiform Anthophyllite	3 Fibers (5.8 % Total Fibers) 3 Fibers (5.8 % Total Fibers)	Date: May 17, 1984
J&J-257	Grantham Ore	Electron Diffraction	Tremolite Chrysotile	0.01-0.1 % 0.001-0.1 %	Preliminary report date: September 3, 1971
J&J-257	Shower to Shower Talcum Powder	Electron Diffraction	Chrysotile	0.001-0.1 %	Preliminary report date: September 3, 1971
J&J-257	Medicated Talcum Powder	Electron Diffraction	Chrysotile	1 Fiber	Preliminary report date: September 3, 1971
J&J-257	Vermont Ore	Electron Diffraction	Asbestos	ND	ND: Not detected Preliminary report date: September 3, 1971

Bates #	Product Tested	Method	Analytes	Results	Comments
J&J-257	Shower to Shower	Electron Diffraction and Microscopy	Asbestiform Minerals	ND	ND: Not detected Final report: "Found three suspect fibers. Of these, two were found in one field and probably have the same source, very possibly contamination"
J&J-257	Medicated Talcum Powder	Electron Diffraction and Microscopy	Asbestiform Minerals	ND	ND: Not detected Final report
J&J-257	Feminine Aerosol Spray	Electron Diffraction and Microscopy	Asbestiform Minerals	ND	ND: Not detected Final report
J&J-257	Vermont Finished Product	Electron Diffraction and Microscopy	Asbestiform Minerals	TR (Tremolite-Actinolite)	TR: Trace # Samples: 7 Results letter date: June 30, 1971
J&J-31	Johnson's Baby Powder	Not Specified	Tremolite Chrysotile	ND ND - 3 %	ND: Not detected # Samples: 3 # Detections: 2 Dr. S. Lewin's (FDA consultant) results Date: September 21, 1972
J&J-31	Johnson's Baby Powder	Not Specified	Tremolite Chrysotile	ND ND - 3 %	ND: Not detected # Samples: 3 # Detections: 2 Dr. S. Lewin's (FDA consultant) results Date: September 21, 1972

Bates #	Product Tested	Method	Analytes	Results	Comments
J&J-31	J&J Medicated Powder	Not Specified	Tremolite Chrysotile	ND - 4 % ND	ND: Not detected # Samples: 2 # Detections: 1 Dr. S. Lewin's (FDA consultant) results Date: September 21, 1972
J&J-31	J&J Shower to Shower	Not Specified	Tremolite Chrysotile	ND 2 %	ND: Not detected # Samples: 3 # Detections: 3 Dr. S. Lewin's (FDA consultant) results Date: September 21, 1972
J&J-31	J&J Shower to Shower	Not Specified	Chrysotile	5%	# Samples: 1 # Detections: 1 Dr. S. Lewin's (FDA consultant) results Date: August 3, 1972
J&J-15	Vermont Talc Product	X-Ray Diffraction	Anthophyllite Tremolite Actinolite	ND <1% <1%	ND: Not detected # Samples: 7 Results letter date: July 7, 1971
J&J-75	Windsor Minerals Talc	Scanning Electron Microscopy and Transmission Electron Microscopy	Asbestos Minerals	~ 1 % (Tremolite) <1% (Possible Chrysotile)	# Samples: 2 # Detections: 2 Results Report Date: February 24, 1975 Report No.: 7690; 1790-79
J&J-175	Windsor Minerals Filter Samples	NIOSH Method P & CAM 239	Asbestos Fibers	<59 - 5,890 Fibers/Filter	# Samples: 4 # Detections: 3 Date: July 14, 1983

Bates #	Product Tested	Method	Analytes	Results	Comments
J&J-97	Talc Ore	Not Specified	Asbestos Fibers	0 - 10 (Amphibole) 1 (Antigorite)	# Samples: 30 # Detections: 10 Date: November 5, 1975
J&J-246	Talc and Rock	Not Specified	Tremolite	4 Detection (Fibrous) 4 Detections (Non-Fibrous)	# Samples: 9
J&J-366	Argonaut Ore Body Ore	Light Microscopy, Transmission Electron Microscopy	Asbestos Minerals	ND - 9 Small Fibers (Chrysotile)	# Samples: 3 # Detections: 1 Report date: May 9, 1974
J&J-366	Argonaut Ore Body Product	Light Microscopy, Transmission Electron Microscopy	Asbestos Minerals	ND - 3 Small Fibers (Chrysotile), 3 Fibers (Possible Chrysotile)	# Samples: 3 # Detections: 1 Report date: May 9, 1974
J&J-335	Johnson's Baby Powder	Petrographic Optical Microscopy	Amphibole	TR (0.001 - 0.1 % Wt)	TR: Trace Dates: October 11, 1972; November 2, 1972; March 9, 1973, March 26, 1973
J&J-305	Talc Powder - Superior Grade EV	Polarized Light Microscopy	Asbestos	2-3% Wt (Tremolite-Actinolite)	Results letter date: January 12, 1984 "It appears to be cleavage fragments of the massive form rather than true asbestiform"
J&J-0144301	Talc	Transmission Electron Microscopy	Serpentine Amphibole	0.0024 % Chrysotile 0.014 % Tremolite	# Samples: 1 Results letter date: March 14, 1988
J&J-0005662	Talc	Transmission Electron Microscopy and Selected Area Electron Diffraction	Serpentine Amphibole	ND	ND: Not detected # Samples: 4 Results letter date: February 18, 1976
JNJ000063951	Johnson's Baby Powder	Optical Microscopy	Asbestos	1 Particle (Tremolite)	Results letter date: October 15, 1995
JNJ000086280	Johnson's Baby Powder	Electron Microscopy	Chrysotile	ND	ND: Not detected Date: July 8, 1973 Reference: 0503.00

Bates #	Product Tested	Method	Analytes	Results	Comments
JNJ000086280	Johnson & Johnson's Green Mountain Talc	Electron Microscopy	Chrysotile	ND	ND: Not detected Date: August 4, 1969 Reference: 0503.00
JNJ000086280	Italian Cosmetic Talc; Talco Grafite	Electron Microscopy	Chrysotile	ND	ND: Not detected Date: June 15, 1973 Reference: 0503.00
JNJNL61_000002060	Cosmetic Grade Chinese Talc	X-Ray Diffraction and Differential Thermal Analysis, Scanning Electron Microscopy and Energy Dispersive X-Ray Analysis	Asbestos	ND	ND: Not detected # Samples: 3 Report Date: March 1983
JNJNL61_000002060	Guping Pit Talc Chips (China	X-Ray Diffraction	Amphibole	ND	ND: Not detected # Samples: 4 Report Date: March 1983
JNJ000222851	Talc Used in Roofing Material	Transmission Electron Microscopy	Asbestiform Minerals	1 Small 2 µm Fiber (Chrysotile)	# Samples: 3 # Detections: 1 Results letter date: August 8, 1974
JNJ000232897	Talc	Electron Diffraction	Asbestiform Minerals	ND - 2 Fibrils (Chrysotile)	ND: Not detected # Samples: 4 # Detections: 1 Results letter date: May 6, 1974
JNJ000223449	Talc	Transmission Electron Microscopy	Asbestos	3 Chrysotile Fibers (10 Grid Squares)	# Samples: 1 # Detections: 1 Results letter date: July 31, 1989

Bates #	Product Tested	Method	Analytes	Results	Comments
JNJ000252742	Talc	Electron Microscopy and Electron Diffraction	Asbestos	ND - 1 Short Fiber (Chrysotile)	ND: Not detected # Samples: 6 # Detections: 1 Results letter date: August 8, 1974
JNJ000260807	Italian Ground Talc	Electron Microscopy	Asbestiform Minerals	ND	ND: Not detected
JNJ000260807	Italian Domestically Ground Talc	Electron Microscopy	Asbestiform Minerals	ND	ND: Not detected
JNJ000281921	Bulk Talc	Transmission Electron Microscopy, Quantitative Adaption of EPA Level II Method, Fiber Identification by Morphology, Energy Dispersive X-Ray Analysis, Selected Area Electron Diffraction	Chrysotile Amphibole	ND ND	ND: Not detected # Samples: 2 Date: August 5, 1992
JNJ000281919	Talc	Transmission Electron Microscopy and X-Ray Diffraction	Asbestos	ND	ND: Not detected # Samples: 2 Results letter date: September 25, 1992
JNJ000314406	Talc Powder	GMP Method and Phase-Contrast Observation	Chrysotile Amphibole	ND ND	ND: Not detected # Samples: 2 Date: February 22, 1977
JNJ00037538	Johnson's Baby Powder	Transmission Electron Microscopy	Asbestos	0.20 % Wt (Anthophyllite)	# Samples: 1 Date Lab Received: December 19, 2003
JNJ000346572	Talc	Transmission Electron Microscopy	Asbestos	ND - 1 3/4 µm Long Particle (Chrysotile)	ND: Not detected # Samples: 2 # Detections: 1 Results letter date: July 17, 1974

Bates #	Product Tested	Method	Analytes	Results	Comments
JNJ000631362	Grade 25 NS	Transmission Electron Microscopy 7024 CTFA J4-1	Asbestos Absence of Asbestos	ND ND	ND: Not detected # Samples: 1 # Detections: 0 Date: August 29, 2016
JNJ000631362	Talc 200J (Cosmetic Grade); China	X-Ray Diffraction and Polarize Light Microscopy	Absence of Asbestos	ND	Date: August 23, 2016
JNJ000631362	Talc Ore; Thailand	Current USP	Absence of Asbestos	Absent	# Samples: 2 Date: 3rd Quarter 2016
JNJ000631362	Talc Ore; India	Current USP	Absence of Asbestos	Absent	# Samples: 1 Date: 4th Quarter 2016
JNJ000631362	Talc Ore Finex; India	Compound Microscopy	Serpentine Tremolite	Absent	# Samples: 1 Date: October 10, 2016
JNJ000631362	Talc Ore Finex; India	X-Ray Diffraction	Asbestiform Tremolite	ND	ND: Not detected # Samples: 1 Date: 4th Quarter 2016
JNJ000631362	Talmag Pharma-S	DRX	Asbestos	ND	ND: Not detected # Samples: 1 Date: April 1, 2016
JNJ000346747	Talc	Transmission Electron Microscopy and Selected Area Electron Diffraction	Asbestiform Minerals	ND	ND: Not detected # Samples: 13 Date: April 5, 1976
JNJ0003447962	Windsor Grade 66	TM7024 (TEM) and CTFA J4-1	Asbestiform Amphibole	ND	# Samples: 1 ND: Not detected
JNJNL61_000006792	Talc	Electron Microscopy	Asbestos	2 Chrysotile Fibers (In 10 Grid Squares) 0 Asbestos Fibers in an Additional 25 Grids	# Samples: 1 Date: May 23, 1989

Bates #	Product Tested	Method	Analytes	Results	Comments
JNJNL61_000027053	Windsor Minerals Ludow 36	X-Ray Diffraction	Anthophyllite Chrysotile Tremolite	ND ND ND	ND: Not detected # Samples: 1 Date Range: July 29-30, 1975
JNJMX68_000013019	Windsor Minerals Talc	Transmission Electron Microscopy	Asbestos	ND - <0.0001 % Wt Chrysotile*	ND: Not detected # Samples: 7 * Because only a few fibers were detected in the portion of each sample analyzed (2 Samples), no accurate value of the weight percent of chrysotile asbestos could be calculated with statistical certainty" Results letter date: August 22, 1985
JNJNL61_000006591	Windsor Mineral Talc	Petrographic Analyses; TEM and Electron Diffraction	Asbestos	ND*	Weekly composite samples # Samples: 49 *"In one instance asbestos was identified...In view of the detected level, 0.006 % and the large number of samples analyzed...more probably represents normal contamination" Results letter date: January 14, 1975
JNJMX68_000004296	Luzenac V-66	Optical Microscopy and Dispersion Staining	Amphibole	ND	ND: Not detected # Samples: 16 Date: 1995
JNJMX68_000004296	Luzenac V-96	Optical Microscopy and Dispersion Staining	Amphibole	ND - 1 Particle ≤ 10 µm	ND: Not detected # Samples: 3 # Detections: 1 Date: 1995

Bates #	Product Tested	Method	Analytes	Results	Comments
JNJNL61_000043271	Windsor Minerals Raw Talc	Transmission Electron Microscopy and Selected Area Electron Diffraction	Asbestiform Minerals	ND	ND: Not detected # Samples: 18 Results letter date: December 2, 1976
JNJNL61_000064161	Windsor Minerals Talc	Not Specified	Asbestiform Minerals	ND	ND: Not detected # Samples: 29 Results letter date: December 31, 1974
JNJS71R_000002199	Grade 66-U-Ore Grade 66 -U-Product	Electron Microscopy	Asbestiform Fiber Count	0 1 Very Small Fiber	Date: May 8, 1973 "Resembled Chrysotile but could not be confirmed"
JNJS71R_000002199	Grade 66-A-Ore Grade 66 -A-Product	Electron Microscopy	Asbestiform Fiber Count	1 Fiber of 8.6 µm length/1.2 µm width 0	Date: May 8, 1973 "Probably Tremolite"
JNJS71R_000002199	Grade 66-AC-Ore Grade 66 -AC-Product	Electron Microscopy	Asbestiform Fiber Count	8 Chrysotile Fibers (Length <1/3 of 1 µm) 1 Chrysotile Fiber	Date: May 8, 1973
IMERYS 045199	Grade 320V	X-Ray Diffraction Polarized Light Microscopy	Serpentine Chrysotile	0.9 % ND	ND: Not detected Silo 5 Composite Date: November 30 - December 1, 1999 Reference: A99543-1
IMERYS 045199	Grade 66	X-Ray Diffraction Polarized Light Microscopy	Serpentine Chrysotile	0.4 % ND	ND: Not detected Silo 2 Composite Date: December 1-6, 1999 Reference: A99543-2
IMERYS 045195	Grade 66	Not Specified	Amphibole	ND	ND: Not detected Silos 2 and 4 Date Milled: June 11 - 22, 1999 and June 22-25, 1999 Reference: A99306

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 430859	Argonaut Drill Samples	X-Ray Diffraction	Serpentine	ND - 0.2 %	ND: Not Detected # Samples: 9 # Detections: 0 Date: October 2, 2002
IMERYS 430859	Argonaut Drill Samples	X-Ray Diffraction	Serpentine	ND - 0.4 %	ND: Not Detected # Samples: 11 # Detections: 4 Date: October 9, 2002
IMERYS 430892	Argonaut Drill Samples	X-Ray Diffraction	Serpentine	ND - 0.8 %	ND: Not Detected # Samples: 26 # Detections: 8 Date: December 20, 2002
IMERYS 430826	Argonaut Drill Samples	X-Ray Diffraction	Serpentine	ND - 1.6* %	ND: Not Detected # Samples: 24 # Detections: 5 *"In cases where a major amount of chlorite is present (~10% +) serpentine calculation may not be accurate" Two Serpentine detections of 1.1% and 1.6% Date: September 30, 2002
IMERYS 430850	Argonaut Drill Samples	X-Ray Diffraction	Serpentine	ND - 11.4 %	ND: Not Detected # Samples: 11 # Detections: 6 Date: November 13, 2002

Bates #	Product Tested	Method	Analytes	Results	Comments
IMERYS 474851	Argonaut Drill Samples	X-Ray Diffraction	Serpentine	ND - 2.9 %	ND: Not Detected # Samples: 10 # Detections: 1 Date: November 13, 2002
IMERYS 475275	Argonaut Drill Samples	X-Ray Diffraction	Serpentine	ND - 80.5 %	ND: Not Detected # Samples: 10 # Detections: 10 Date: February 28, 2003
IMERYS 475372	Float Feed	X-Ray Diffraction	Serpentine	ND	ND: Not detected Silo 9 (32 hrs.) Date: January 15-16, 2003 Reference: A03057-1
IMERYS 475372	Float Feed	X-Ray Diffraction	Serpentine	1.70%	Date: January 17, 2003 Reference: A03057-2
IMERYS 475275	Argonaut Drill Samples	X-Ray Diffraction	Serpentine	ND - 0.8 %	ND: Not Detected # Samples: 24 # Detections: 10 Date: February 6, 2003
IMERYS 481989	Argonaut Drill Samples	X-Ray Diffraction	Serpentine	ND	ND: Not Detected # Samples: 20 # Detections: 0 Date: February 18, 2003

Bates #	Product Tested	Method	Analytes	Results	Comments
JOJO-MA2330-0033	Johnson's Baby Powder	Optical and Electron Microscopy	Asbestos Asbestiform Minerals	ND ND	ND: Not detected # Samples: 1 Batch 344L Results letter date: August 19, 1971

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
IMERYS 045185	Talc	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Cadmium Cobalt Chromium Nickel Arsenic Lead	<0.24 mg/kg 81.9 mg/kg 136 mg/kg 2,180 mg/kg 0.3 mg/kg 1.5 mg/kg	Grade 66 yearly composite sample: 1999 Reference: A99063
IMERYS 045183	Talc	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Cadmium Cobalt Chromium Nickel Arsenic Lead	<0.2 mg/kg 76.8 mg/kg 199 mg/kg 2,000 mg/kg 0.6 mg/kg 4.7 mg/kg	Grade 66 (High Insol): 5/23/00 Reference: A00470-1
IMERYS 045183	Talc	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Cadmium Cobalt Chromium Nickel Arsenic Lead	<0.2 mg/kg 77.3 mg/kg 209 mg/kg 1,960 mg/kg 1.2 mg/kg 1.7 mg/kg	Grade 66 (Mid Insol): 5/23/00 Reference: A00470-2
IMERYS 045183	Talc	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Cadmium Cobalt Chromium Nickel Arsenic Lead	<0.2 mg/kg 76.8 mg/kg 324 mg/kg 1,890 mg/kg 1.5 mg/kg 0.7 mg/kg	Grade 66 (Low Insol): 5/23/00 Reference: A00470-3
IMERYS 053276	Grade 66	TM7717 QA-019 (USP) TM7168	Arsenic Heavy Metals (Lead ≤ 10 ppm)	<2.5 ppm Passed	Grade 66 Silo 4 Batch # 1065-1075 Production Period: March 22 - April 3, 1996 Luzenac America certifies that this product does not contain asbestos
IMERYS 053277	Grade 66	TM7717 QA-019 (USP) TM7168	Arsenic Heavy Metals (Lead ≤ 10 ppm)	<2.5 ppm Passed	Grade 66 Silo 5 Batch # 1075-1081 Production Period: April 3 - 11, 1996 Luzenac America certifies that this product does not contain asbestos
IMERYS 053387	Grade 66	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Cadmium Cobalt Chromium Nickel Arsenic Lead	0.4 mg/kg 76.2 mg/kg 262 mg/kg 2,100 mg/kg	Grade 66 Low Insolubles Study WO01018-xxx Comp (16A, 18A, 19A, 20A) Results letter date: February 21, 2001 Reference: A01066

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
IMERYS 058045 IMERYS 058046	Powder	EP 04/2005:0438 Talc Monograph FCC V Talc Monograph HTM_059 USP 30 - Talc Monograph FCC V Talc Monograph	Lead Arsenic	<0.001 % <5 mg/kg 0.5 ppm (Wt) <0.001 Wt % <3 mg/kg	Sample ID: 2007-006529-DRPK-001 Sample Description: GUANGXI 1 Date Sampled: 6/1/2007
IMERYS 058047 IMERYS 058048	Powder	EP 04/2005:0438 Talc Monograph FCC V Talc Monograph HTM_059 USP 30 - Talc Monograph FCC V Talc Monograph	Lead Arsenic	<0.001 % <5 mg/kg 0.2 ppm (Wt) <0.001 Wt % <3 mg/kg	Sample ID: 2007-006529-DRPK-002 Sample Description: GUANGXI 2 Date Sampled: 6/1/2007
IMERYS 058049 IMERYS 058050	Powder	EP 04/2005:0438 Talc Monograph FCC V Talc Monograph HTM_059 USP 30 - Talc Monograph FCC V Talc Monograph	Lead Arsenic	<0.001 % <5 mg/kg 0.2 ppm (Wt) <0.001 Wt % <3 mg/kg	Sample ID: 2007-006529-DRPK-003 Sample Description: GUANGXI 3 Date Sampled: 6/1/2007
IMERYS 058069	Ore	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	<0.2 ppm <0.02 ppm 0.1 ppm 1 ppm <0.2 ppm <0.5 ppm	Crude ore sample of Guangxi #2: MV Makali RM Composite Sample: May 2007 Reference: A07138-1
IMERYS 058069	Ore	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	<0.2 ppm <0.02 ppm 2.4 ppm 1 ppm 3.1 ppm 0.7 ppm	Crude ore sample of Guangxi #2: MV Makali ACM Composite Sample: May 2007 Reference: A07138-2
IMERYS 058069	Ore	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	<0.2 ppm <0.02 ppm 2.3 ppm 1 ppm 2.6 ppm 0.5 ppm	Crude ore sample of Guangxi #2: MV Makali AFG Composite Sample: May 2007 Reference: A07138-3
IMERYS 058072	Ore	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	<0.2 ppm <0.02 ppm 1.8 ppm 1 ppm 2.5 ppm 0.6 ppm	Crude ore sample of Guangxi #2A: MV Makali RM Composite Sample: June 2007 Reference: A07136-1

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
IMERYS 058072	Ore	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	<0.2 ppm <0.02 ppm 1.9 ppm <1 ppm 2.6 ppm 0.9 ppm	Crude ore sample of Guangxi #2A: MV Makali ACM Composite Sample: June 2007 Reference: A07136-2
IMERYS 058072	Ore	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	0.2 ppm <0.02 ppm 1.9 ppm 1 ppm 2.7 ppm 0.6 ppm	Crude ore sample of Guangxi #2A: MV Makali AFG Composite Sample: June 2007 Reference: A07136-3
IMERYS 058220	Talc	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	<0.2 ppm <0.02 ppm 2.7 ppm 4 ppm 4.6 ppm 1.1 ppm	Sample: RM Guangxi2 M.V. Blunenau: December 2009 Reference: A10043-1
IMERYS 058220	Talc	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	1.2 ppm <0.02 ppm 1.6 ppm 3 ppm 2.7 ppm 0.5 ppm	Sample: ACM Guangxi2 M.V. Blunenau: December 2009 Reference: A10043-2
IMERYS 058220	Talc	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	0.63 ppm <0.02 ppm 2.4 ppm 3 ppm 4.3 ppm 0.5 ppm	Sample: AFG Guangxi2 M.V. Blunenau: December 2009 Reference: A10043-3
IMERYS 058228 IMERYS 058229	Talc	FCC VI ITM 1059	Arsenic Lead Arsenic Cadmium Chromium Nickel Lead	<3 mg/kg <5 mg/kg <1.00 ppm Wt <1.00 ppm Wt <0.500 ppm Wt <1.00 ppm Wt <1.00 ppm Wt	Sample ID: 2010-DRPK-000997-001 Representing: M.V. Blunenau Grind Date: 10/06/09

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
IMERYS 058231 IMERYS 058232	Talc	EP 6.01 USP 31 - NF 26 FCC VI ITM 1059	Lead Lead Arsenic Lead Arsenic Cadmium Chromium Nickel Lead	1.2 ppm 1.240 % <3 mg/kg <5 mg/kg <1.00 ppm Wt <1.00 ppm Wt <0.500 ppm Wt <1.00 ppm Wt <1.00 ppm Wt	Sample ID: 2009-DRPK-011976-001 Representing: Guangxi 2 (M.V. Beilun Dolphin) Grind Date: 10/06/09
IMERYS 113412 IMERYS 113413	Talc	United States Pharmacopeia British Pharmacopeia European Pharmacopeia Food Chemicals Codex Japanese Pharmacopeia	Arsenic Heavy Metals Lead	<3 ppm <40 ppm <10 - 1.2 ppm	Sample: Guangxi #2A Grind Date: 2/18/04 Ship Name: Tequi V. 34011 pt #580
IMERYS 113424 IMERYS 113425	Talc	United States Pharmacopeia British Pharmacopeia European Pharmacopeia Food Chemicals Codex Japanese Pharmacopeia	Arsenic Heavy Metals Lead	<3 ppm <40 ppm <10 - 2.63 ppm	Sample: Guangxi #1 Date: January 2004 Pre-Shipment MV Tequi
IMERYS 113426 IMERYS 113427	Talc	United States Pharmacopeia British Pharmacopeia European Pharmacopeia Food Chemicals Codex Japanese Pharmacopeia	Arsenic Heavy Metals Lead	<3 ppm <40 ppm <10 - 3.43 ppm	Sample: Guangxi #2A Date: January 2004 Pre-Shipment MV Tequi
IMERYS 113430 IMERYS 113431	Talc	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	0.7 ppm <0.02 ppm 2.3 ppm 2 ppm 4 ppm 0.6 ppm	Sample: Houston February 2004 Guangxi 2A - RM MV Tequi Reference: A04112-1
IMERYS 113430 IMERYS 113431	Talc	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	0.3 ppm <0.02 ppm 2.3 ppm 2 ppm 4.1 ppm <0.5 ppm	Sample: Houston February 2004 Guangxi 2A - ACM MV Tequi Reference: A04112-2
IMERYS 113430 IMERYS 113431	Talc	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	<0.2 ppm <0.02 ppm 2.1 ppm 2 ppm 3.7 ppm <0.5 ppm	Sample: Houston February 2004 Guangxi 2A - AFG MV Tequi Reference: A04112-3

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
IMERYS 113433 IMERYS 113434	Talc	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	0.4 ppm <0.02 ppm 1.4 ppm 1 ppm 2.1 ppm <0.5 ppm	Sample: Houston February 2004 Guangxi #1 - RM MV Tequi Reference: A04113-1
IMERYS 113433 IMERYS 113434	Talc	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	0.4 ppm <0.02 ppm 1.5 ppm 3 ppm 2.6 ppm <0.5 ppm	Sample: Houston February 2004 Guangxi #1 - ACM MV Tequi Reference: A04113-2
IMERYS 113433 IMERYS 113434	Talc	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	<0.2 ppm <0.02 ppm 1.4 ppm 1 ppm 2.1 ppm <0.5 ppm	Sample: Houston February 2004 Guangxi #1 - AFG MV Tequi Reference: A04113-3
IMERYS 113570 IMERYS 113571	Talc	USP 31 - NF 26 EP 6.01 FCC V ITM 1059	Lead Lead Arsenic Lead Lead	<0.001 % 2.9 ppm <3 mg/kg <5 mg/kg 1.77 ppm Wt	Sample ID: 2009-DRPK-000398-001 Representing: Guangxi 1: January 2009
IMERYS 113570 IMERYS 113571	Talc	USP 31 - NF 26 EP 6.01 FCC V ITM 1059	Lead Lead Arsenic Lead Lead	<0.001 % 4.3 ppm <3 mg/kg <5 mg/kg 1.99 ppm Wt	Sample ID: 2009-DRPK-000398-002 Representing: Guangxi 1: January 2009
IMERYS 113578 IMERYS 113579 IMERYS 113580	Ore	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	0.3 ppm <0.02 ppm 2.2 ppm 3 ppm 3 ppm 1 ppm	Crude ore sample of Guangxi #2 RM: MV Anna Smile Sample: November 2008 Reference: A09015-1
IMERYS 113578 IMERYS 113579 IMERYS 113580	Ore	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	0.3 ppm <0.02 ppm 2.2 ppm 4 ppm 3.3 ppm 0.9 ppm	Crude ore sample of Guangxi #2 ACM: MV Anna Smile Sample: November 2008 Reference: A09015-2

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
IMERYS 113578 IMERYS 113579 IMERYS 113580	Ore	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	<0.2 ppm <0.02 ppm 2.2 ppm 2 ppm 2.8 ppm 0.8 ppm	Crude ore sample of Guangxi #2 AFG: MV Anna Smile Sample: November 2008 Reference: A09015-3
IMERYS 113578 IMERYS 113579 IMERYS 113580	Ore	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	0.4 ppm <0.02 ppm 1.3 ppm 2 ppm 2 ppm 1.5 ppm	Crude ore sample of Guangxi #1 RM: MV Anna Smile Sample: November 2008 Reference: A09015-4
IMERYS 113578 IMERYS 113579 IMERYS 113580	Ore	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	0.4 ppm <0.02 ppm 1.6 ppm 2 ppm 2.5 ppm 1.6 ppm	Crude ore sample of Guangxi #1 ACM: MV Anna Smile Sample: November 2008 Reference: A09015-5
IMERYS 113578 IMERYS 113579 IMERYS 113580	Ore	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	0.3 ppm <0.02 ppm 1.5 ppm 2 ppm 2.4 ppm 1.9 ppm	Crude ore sample of Guangxi #1 AFG: MV Anna Smile Sample: November 2008 Reference: A09015-6
IMERYS 113607 IMERYS 113608	Talc	USP 31 - NF 26 EP 6.01 FCC V ITM 1059	Lead Lead Arsenic Lead Lead	1.240 % 1.2 ppm <3 mg/kg <5 mg/kg <1.00 ppm Wt	Sample ID: 2009-DRPK-011976-001 Representing: Guangxi 1: October 2009
IMERYS 113614 IMERYS 113615	Talc	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	<0.2 ppm <0.02 ppm 2.5 ppm 5 ppm 4.8 ppm 0.6 ppm	Sample: RM Guangxi2 M.V. Blunenau: October 21, 2009 Reference: A09479-1
IMERYS 113614 IMERYS 113615	Talc	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	<0.2 ppm <0.02 ppm 2.8 ppm 3 ppm 4.9 ppm 0.6 ppm	Sample: ACM Guangxi2 M.V. Blunenau: October 21, 2009 Reference: A09479-2

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
IMERYS 113614 IMERYS 113615	Talc	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	<0.2 ppm <0.02 ppm 2.8 ppm 3 ppm 4.5 ppm 0.5 ppm	Sample: AFG Guangxi2 M.V. Blunenau: October 21, 2009 Reference: A09479-3
IMERYS 225184	Talc	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Cadmium Cobalt Chromium Nickel Arsenic Lead	<0.2 mg/kg 76.8 - 77.3 mg/kg 199 - 324 mg/kg 1,890 - 2,000 mg/kg 0.6 - 1.5 mg/kg 0.7 - 4.7 mg/kg	# Samples: 3 Date: 2000
IMERYS 225295	Talc	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Arsenic Cadmium Cobalt Chromium Nickel Lead	<2.50 mg/kg <0.20 mg/kg <0.60 mg/kg 0.81 mg/kg <1.0 mg/kg <0.8 mg/kg	Date: April 12, 2006
IMERYS 286445	Talc	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Arsenic Cadmium Cobalt Chromium Nickel Lead	2.63 mg/kg 0.26 mg/kg 77.3 mg/kg 284 mg/kg 2,100 mg/kg 1.11 mg/kg	Grade 96 USP yearly composite sample: 2003 Reference: A03099
IMERYS 304036	Grade 66	Inductively Coupled Plasma Chemical Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	10 - 19 ppm <0.5 ppm 79 - 89 ppm 230 - 288 ppm 2,410 - 2,510 ppm 4 - 6 ppm	# Samples: 3 Date: May 11, 1998 (1 Sample) and September 24, 1998 (2 Samples) Reference: A00600-1, A00600-2, A00600-3
IMERYS 340798	Grade 96 USP	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Arsenic Cadmium Cobalt Chromium Nickel Lead	0.328 mg/kg 0.22 mg/kg 71.3 mg/kg 245 mg/kg 1,980 mg/kg 0.165 mg/kg	Yearly composite sample: 2002 Reference: A02225

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
IMERYS 340454	Grade 66	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Arsenic Cadmium Cobalt Chromium Nickel Lead	<0.5 mg/kg 0.5 mg/kg 79.6 mg/kg 223 mg/kg 2,260 mg/kg <0.25 mg/kg	Yearly composite sample: 1999 Reference: A01188
IMERYS 342524	Grade 66	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Arsenic Cadmium Cobalt Chromium Nickel Lead	0.7 mg/kg <0.24 mg/kg 8.1 mg/kg 25.4 mg/kg 247 mg/kg 0.4 mg/kg	Yearly composite sample: 1997 Reference: 97-024 (Recheck)
IMERYS 342524	Grade 66	Inductively Coupled Plasma Chemical Analysis: Triple Acid Total Digestion	Arsenic Cadmium Cobalt Chromium Nickel Lead	2 ppm <0.5 ppm 92 ppm 273 ppm 2,490 ppm <2 ppm	Yearly composite sample: 1997 Reference: 97-024 (Recheck)
IMERYS 427237	Rainbow Mine Whole Rock	Atomic Absorption	Arsenic	79.6 - 83.3 ppm	Date: September 6, 1990 # Samples: 5 All samples from 1270 Level
IMERYS 427237	Rainbow Mine Sludge	Atomic Absorption	Arsenic	42.3 - 172.3 ppm	Date: August 29, 1990 # Samples: 9
IMERYS 427238	Rainbow Mine Core	Atomic Absorption	Arsenic	0.83 - 8.72 ppm	Date: September 19, 1990 # Samples: 6
IMERYS 427238	Rainbow Mine Core	Atomic Absorption	Arsenic	0.09 - 3.10 ppm	Date: September 20, 1990 # Samples: 7
IMERYS 427239	Argonaut Mine Whole Rock	Atomic Absorption	Arsenic	0.02 - 12.78 ppm	Date: November 16, 1990 # Samples: 15
IMERYS 427244 IMERYS 427245 IMERYS 427246 IMERYS 427247	Rainbow Ore	Not Specified	Arsenic	0.3 - 158.3 *Units not specified*	Arsenic analysis: 1990 Arsenic program for Rainbow Mine # Samples: 169

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
IMERYS-A_0002017	Vermont Talc Ore	Atomic Absorption and Inductively Coupled Plasma Chemical Analysis EPA 3060A/7199 for Hexavalent Chromium	Lead Cadmium Nickel Chromium Hexavalent Chromium Cobalt Arsenic	2 ppm 3 ppm 1,685 ppm 1,700 ppm <4 ppb 88 ppm 65 ppm	Results letter date: November 2006
IMERYS-A_0015663	Grade 66	X-Ray Fluorescence	Cadmium Cobalt Chromium Nickel	<0.24 mg/kg 67.4 - 82.9 mg/kg 85.8 - 169 mg/kg 1,810 - 2,190 mg/kg	Grade 66 Non-Shear Disc Test Run # Samples: 5 Dates: 3/25/1998, 5/11/1998, 9/24/1998, 9/25/1998, 8/18-21/1999 Reference: A99372
IMERYS-MDL-AB_0005581 IMERYS-MDL-AB_0005582 IMERYS-MDL-AB_0005583 IMERYS-MDL-AB_0005584	Cyprus Industrial Minerals Pulp	Not Specified	Total Lead Extractable Lead Total Nickel Extractable Nickel Total Chromium Extractable Chromium Total Arsenic Extractable Arsenic	<2 - 48 ppm <1 - 3 ppm 95 - 1,050 ppm 13 - 65 ppm 210 - 5,550 ppm <1 - 3 ppm <2 - 570 ppm <1 - 22 ppm	# Samples: 47 Report Date: April 12, 1988
J&J-0083336	Grade 66 U	Not Specified	Arsenic Heavy Metals	0.13 ppm <10 ppm	Ultrawet D.S. floated product Date: 1/29/74
J&J-0083337	Grade 66 A	Not Specified	Arsenic Heavy Metals	0.07 ppm <10 ppm	N-butanol floated product Date: 1/29/74
J&J-0083338	Grade 66 AC	Not Specified	Arsenic Heavy Metals	0.17 ppm <10 ppm	N-butanol, citric acid floated Date: 1/29/74
JNJ000087928	J&J 228P (Baby Powder)	Not Specified	Arsenic Lead Nickel Cobalt Chromium	0.4 ppm 0.0 ppm 1,300 ppm 0.0 ppm 100.0 ppm	Date: October 1972

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
JNJ000088570	Talc	Atomic Absorption Analyses by J&J Digestion Procedure	Cobalt Chromium Nickel	63.6 - 84.4 ppm Wt 194 - 305 ppm Wt 2,090 - 2,650 ppm Wt	# Samples: 3 # Detections: 9 Results Date: February 12, 1981 Reference: 1495
JNJ000063608	Talc	Method 7061 Method 7130 Method 7200 Method 7190 Method 7420 Method 7520	Arsenic Cadmium Cobalt Chromium Lead Nickel	1.09 mg/kg ND 60 mg/kg 569 mg/kg BDL 2,070 mg/kg	ND: Not detected BDL: Below detection limit Report Date: March 13, 1995
JNJNL61_000002060	Cosmetic Grade Chinese Talc	Metal Oxide Analysis	Arsenic	<2 ppm	# Samples: 3 Report Date: March 1983
JNJNL61_000002060	Cosmetic Grade Chinese Talc	Trace Metals by Atomic Absorption	Nickel Chromium Cobalt	5 - 6 ppm 4 - 5 ppm 9 - 10 ppm	# Samples: 3 Report Date: March 1983
JNJNL61_000002060	Guping Pit Talc Chips (China	Trace Metals by Atomic Absorption	Nickel Chromium Cobalt Lead Arsenic	15 ppm 1.6 ppm 7.5 ppm 12.7 ppm 1 ppm	# Samples: 1 Composite Report Date: March 1983
JNJ000237076	Talc	USP XXII Talc Heavy Metals Method Mod. For A.A. J&J Method BPT-148 USP XXII Talc Heavy Metals Method Mod. For A.A. J&J Method BPT-148	Arsenic Cadmium Chromium Cobalt Lead Nickel	1.35 - 1.54 mg/kg ND 251 - 277 mg/kg 56 - 57 mg/kg 0.05 - 0.06 mg/kg 1,720 - 1,942 mg/kg	ND: Not detected # Samples: 2 Report Date: October 1, 1991
JNJ000239730	Talc	Method 7061 Method 7130 Method 7200 Method 7190 Method 7420 Method 7520	Arsenic Cadmium Cobalt Chromium Lead Nickel	1.21 mg/kg ND 67 mg/kg 457 mg/kg 0.14 mg/kg 2,260 mg/kg	ND: Not detected Report Date: March 10, 1994
JNJ000237279	Windsor Talc	Atomic Absorption	Nickel	0.19 - 0.25 *Units not specified*	# Samples: 2 Results letter date: December 31, 1975

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
JNJ000238011	Johnson's Baby Powder	Atomic Absorption	Nickel Cobalt Chromium	1,480 - 1,500 ppm ND - 57 ppm ND - 190 ppm	ND: Not detected # Samples: 3 Results letter date: September 30, 1976 Reference: 0503.01
JNJ000246437	Talc	Not Specified	Cadmium Chromium Cobalt Lead Nickel	ND 7 - 426 mg/kg 17 - 83 mg/kg ND 31 - 1,940 mg/kg	ND: Not detected # Samples: 3 Results letter date: February 7, 1990 Reference: 017889
JNJ000239723	Talc	J&J Method BPT 148 and TM 7165	Arsenic Cadmium Chromium Cobalt Lead Nickel	1.52 mg/kg ND 328 mg/kg 63 mg/kg 0.07 mg/kg 2,100 mg/kg	ND: Not detected # Samples: 1 Date: April 27, 1992
JNJ000246467	Johnson's Baby Powder	Not Specified	Nickel Cobalt Chromium	2,050 ppm 66 ppm 200-400 ppm	ND: Not detected # Samples: 1 Results letter date: September 15, 1976 In talc lattice; Lot 228P
JNJ000246467	Johnson's Baby Powder	Not Specified	Nickel Cobalt Chromium	30 ppm 4 ppm "A Few ppm"	ND: Not detected # Samples: 1 Results letter date: September 15, 1976 In non-talc components; Lot 228P
JNJ000291914	Grade 66	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Cadmium Cobalt Chromium Nickel Arsenic Lead	<0.24 mg/kg 8.1 mg/kg 25.4 mg/kg 247 mg/kg 0.7 mg/kg 0.4 mg/kg	Date: 1996 yearly composite

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
JNJ000285351	Talc	Not Specified	Cadmium Chromium Cobalt Lead Nickel	3 262 - 294 84 - 85 40 - 41 2,450 - 2,560 *Units Not Specified*	# Samples: 1 # Duplicate Samples: 1 Date: December 19, 1988
JNJ000291916	Grade 66	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Cadmium Cobalt Chromium Nickel Arsenic Lead	<0.5 mg/kg 77.9 mg/kg 255 mg/kg 2,060 mg/kg <0.2 mg/kg 6.10 mg/kg	Date: 1997 yearly composite
JNJ000631362	Grade 25 NS	ICP-MS	Arsenic Cadmium Chromium Nickel	<1 ppm <0.5 ppm <0.5 ppm <0.5 ppm	# Samples: 1 # Detections: 0 Date: August 29, 2016
JNJ000631362	Talc Ore; Thailand	Current USP BIS 1462 AA	Lead Arsenic Cadmium Chromium Nickel	0.55 - 0.61 ppm <2 ppm <0.01 ppm 0.25 - 0.27 ppm <0.08 ppm	# Samples: 2 Date: 3rd Quarter 2016
JNJ000631362	Talc Finex; India	Not Specified	Arsenic Heavy Metals	< 2 ppm < 10 ppm	# Samples: 1 Date: January 11, 2016
JNJ000631362	Talc Ore Finex; India	Current USP BIS 1462 AA	Lead Arsenic Cadmium Chromium Nickel	<10 ppm <2 ppm <0.01 ppm 0.25 ppm <0.08 ppm	# Samples: 1 Date: 4th Quarter 2016
JNJ000631362	Talc Ore Finex; India	IS: 1462-1985	Heavy Metals	<10 ppm	# Samples: 1 Date: October 10, 2016
JNJ000631362	Talc Ore Finex; India	USP	Cadmium Nickel Chromium	<0.01 ppm <0.08 ppm 0.25 ppm	# Samples: 1 Date: October 12, 2016
JNJ000631362	Talmag Pharma-S	EE Plasma USP/EP/JP	Heavy Metals (Max) Lead (Max) Arsenic (Max)	40.0 ppm 0.001 % 4.0 ppm	ND: Not detected # Samples: 1 Date: April 1, 2016

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
JNJ000886067	Talc	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81)	Cadmium Cobalt Chromium Nickel Arsenic Lead	<0.1 mg/kg 72.9 mg/kg 275 mg/kg 1.7 mg/kg 1.4 mg/kg	# Samples: 1 Date: February 9, 1999 Reference: A98015
JNJ0003447962	Non-Shear Disc Talc	Johnson & Johnson Test No BPT 148 ("Determination of Trace Elements in Talc by Atomic Absorption," Date: 11/23/81); TM169, CTFA USP	Cadmium Cobalt Chromium Nickel Arsenic Lead	<0.24 mg/kg 67.4 - 79.3 mg/kg 85.8 - 110 mg/kg 2,020 - 2,190 mg/kg 0.66 - 0.67 ppm 13.8 - 14.5 mg/kg	# Samples: 3
JNJ000378046	Talc	EPA 7199	Hexavalent Chromium	ND - 70 µg/kg	ND: Not detected # Samples: 4 # Detections: 1 Date: April 28, 2010
IMERYS 430859	Argonaut Drill Samples	ICP Analysis	Lead Nickel Cobalt Cadmium Arsenic Chromium	<2 - 5 ppm 1,162 - 1,851 ppm 58 - 86 ppm <1 ppm <5 - 25 ppm 1,033 - 2,505 ppm	ND: Not Detected # Samples: 17 Date: October 2, 2002
IMERYS 430859	Argonaut Drill Samples	ICP Analysis	Lead Nickel Cobalt Cadmium Arsenic Chromium	<2 - 6 ppm 851 - 1,812 ppm 62 - 92 ppm <1 - 3 ppm <5 - 234 ppm 1,311 - 3,215 ppm	ND: Not Detected # Samples: 25 Date: October 9, 2002
IMERYS 430892	Argonaut Drill Samples	ICP Analysis	Lead Nickel Cobalt Cadmium Arsenic Chromium	<2 - 8 ppm 1,004 - 1,772 ppm 54 - 87 ppm <1 ppm <5 - 42 ppm	ND: Not Detected # Samples: 26 Date: December 20, 2002

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
				1,143 - 2,332 ppm	
IMERYS 430826	Argonaut Drill Samples	ICP Analysis	Lead Nickel Cobalt Cadmium Arsenic Chromium	<2 - 14 ppm 1,443 - 1,900 ppm 77 - 90 ppm <1 ppm <5 - 135 ppm 2,122 - 8,500 ppm	ND: Not Detected # Samples: 24 Date: September 30, 2002
IMERYS 430850	Argonaut Drill Samples	ICP Analysis	Lead Nickel Cobalt Cadmium Arsenic Chromium	<2 - 4 ppm 979 - 1,896 ppm 74 - 88 ppm <1 ppm <5 - 235 ppm 1,543 - 4,912 ppm	ND: Not Detected # Samples: 16 Date: October 2, 2002
IMERYS 430850	Argonaut Drill Samples	ICP Analysis	Lead Nickel Cobalt Cadmium Arsenic Chromium	<2 - 5 ppm 1,762 - 2,042 ppm 85 - 96 ppm <1 ppm <5 - 159 ppm 980 - 1,761 ppm	ND: Not Detected # Samples: 11 Date: November 13, 2002
IMERYS 474851	Argonaut Drill Samples	ICP Analysis	Lead Nickel Cobalt Cadmium Arsenic Chromium	<2 - 4 ppm 1,357 - 1,837 ppm 81 - 91 ppm <1 ppm <5 - 9 ppm 1,721 - 2,352 ppm	ND: Not Detected # Samples: 10 Date: November 13, 2002
IMERYS 475275	Argonaut Drill Samples	ICP Analysis	Lead Nickel Cobalt Cadmium Arsenic Chromium	<2 - 4 ppm 1,375 - 1,938 ppm 80 - 95 ppm <1 ppm <5 ppm 1,608 - 3,203 ppm	ND: Not Detected # Samples: 12 Date: February 28, 2003

Bates #	Product Tested	Method (if applicable)	Analytes	Results	Comments
IMERYS 475372	Float Feed	X-Ray Diffraction and Inductively Coupled Plasma Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	2.8 ppm <0.02 ppm 79.7 ppm 171 ppm 2,120 ppm 1.7 ppm	ND: Not detected Silo 9 (32 hrs.) Date: January 15-16, 2003 Reference: A03057-1
IMERYS 475372	Float Feed	X-Ray Diffraction and Inductively Coupled Plasma Analysis	Arsenic Cadmium Cobalt Chromium Nickel Lead	1.0 ppm <0.02 ppm 84.5 ppm 1,610 ppm 1,600 ppm 0.7 ppm	Date: January 17, 2003 Reference: A03057-2
IMERYS 475275	Argonaut Drill Samples	ICP Analysis	Lead Nickel Cobalt Cadmium Arsenic Chromium	<2 - 30 ppm 1,262 - 1,866 ppm 65 - 91 ppm <1 ppm <5 ppm 1,466 - 2,561 ppm	ND: Not Detected # Samples: 24 Date: February 6, 2003
IMERYS 481989	Argonaut Drill Samples	ICP Analysis	Lead Nickel Cobalt Cadmium Arsenic Chromium	<2 - 10 ppm 1,019 - 1,950 ppm 70 - 87 ppm <1 ppm <5 - 478 ppm 872 - 2,016 ppm	ND: Not Detected # Samples: 20 Date: February 18, 2003

APPENDIX D

Attorney's Eyes Only – Fragrant Chemical Information

REDACTED

ATTORNEYS' EYES ONLY

APPENDIX E

Rule 26 Testifying History of Dr. Scribner Tuttle

CTEH®
THE SCIENCE OF READY℠

Previous 4 Years of Expert Testimony
Kelly Scribner Tuttle, Ph.D., CIH

In the Superior Court of the State of California for the County of Los Angeles
Baukholt v A.O. Smith et al.
No. BC599035
Deposition Testimony June 24, 2016

In the District Court, Eighth Judicial District
Thomas Wilson v Eighty-Eight Oil
No. CV-2014-127-DC
Trial Testimony December 9, 2016

In the Superior Court of Washington for King County
Readwin v Aurora Pumps, et al.
No. 16-2-18894-0 SEA
Deposition Testimony February 15, 2017

In the 27th Judicial District Court, Parish of St. Landry, State of Louisiana
Joshua Adams et al. v. Union Pacific Railroad Company
No. 14-C-3165-B
Trial Testimony June 28, 2017

In the District Court, Rockwall County, Texas 439th Judicial District
Hudson v Covestro
No. 1-15-848
Deposition Testimony July 21, 2017

In the 27th Judicial District Court In and For the Parish of St. Landry, State of Louisiana
Andrus, Jones, Adams, Shakesnider and White et al. v Union Pacific Railroad
No. 14-C-3164-A, 14-C-3164-B, 14-C-3164-C, 14-C-3164-D
Trial Testimony August 15 & 17, 2017

In the 27th Judicial District Court In and For the Parish of St. Landry, State of Louisiana
Andrus, Jones, Adams, Shakesnider and White et al. v Union Pacific Railroad
No. 14-C-3164-A, 14-C-3164-B, 14-C-3164-C, 14-C-3164-D
Trial Testimony October 10, 2017

In the United States District Court, Northern District of Illinois, Eastern Division
Walter Messel v Aurora Pump, et al.
No. 14-C-3164-A, 14-C-3164-B, 14-C-3164-C, 14-C-3164-D
Deposition Testimony November 11, 2017

In the Court of Common Pleas, State of South Carolina, County of York
Wayne Howe v Aurora Pump, et al.
No. 2015-CP-46-3456
Deposition Testimony February 26, 2018

In the Superior Court of the State of California for the County of Alameda
Charles Bradd v Aurora Pump, et al.
No. RG17884478
Deposition Testimony June 29, 2018

State of Texas, Workers Comp Court
Linda Malone v. SORM
Hearing Testimony November 27, 2018